JOURNAL
of the
American Veterinary Medical Association
Formerly AMERICAN VETERINARY REVIEW
(Original Official Organ U. S. Vet. Med. Assn.)
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Vol. LXXXV, N. S. Vol. 38 AUGUST, 1934 No. 2

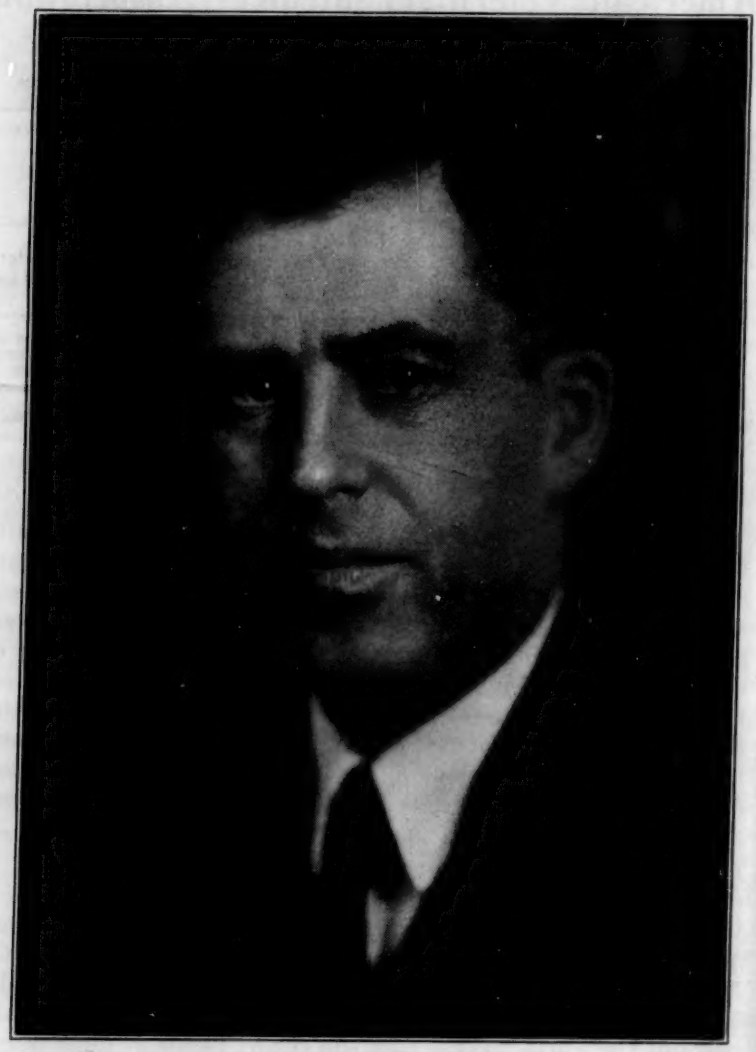
THE CONGRESS DRAWS NEAR

The event toward which veterinarians in all parts of the world have been looking for several years is now only a few days away—the Twelfth International Veterinary Congress. To say that it will be the largest gathering of veterinarians ever assembled is a reasonably safe prediction. That the enrollment of members will surpass any previous Congress is already assured. Even the threat of war in Europe and the financial difficulties of many foreign countries have not dampened the enthusiasm for the Congress, although in numerous instances it has been difficult, if not impossible, to remit funds for membership fees.

It has been a genuine pleasure to read the letters received from veterinarians in more than fifty foreign countries and to sense the high value which these men attach to membership in the Congress. Many who will be unable to be present in New York will eagerly await the reports, so that they may read of the latest developments in the field of veterinary science, revealed by more than eighty reporters chosen from the profession in all parts of the world. These men will report on about forty different subjects, practically covering the entire field of veterinary medicine and surgery.

It has been a source of considerable gratification to note the interest in the Congress that is being taken by members of the

To Open Congress



HON. HENRY A. WALLACE
Secretary of Agriculture

medical profession. The constantly growing interest in the intercommunicability of certain infectious diseases undoubtedly is responsible for a great share of this interest in the Congress. The professions of veterinary medicine and human medicine are so closely related that it would be impossible to separate them. Each one can profit by what the other has to offer, and mankind will be the chief beneficiary of this free, unselfish interchange of scientific knowledge.

Other important fields of endeavor are being attracted to the Congress. The very fact that Secretary of Agriculture Wallace has promised to open the Congress is in itself significant of the importance of the event to agriculture. The packing industry will watch the deliberations of the Congress very closely, as the movement of dressed meats and meat food products between this and other countries is directly affected by live stock diseases and methods for their control. Transportation companies will have their representatives present, as will the dairy industry, to get first-hand information concerning any developments in veterinary medicine that will affect their business. All of which goes to emphasize the importance of and the strategic position occupied by veterinary science.

Practically every veterinarian in the United States has had at least one invitation to become a member of the Congress. Many have been asked more than once. Very few have been missed. A year ago it was estimated that we could count on at least 3,000 members from this country. We are quite a way from that mark as this is written, but there is still time to reach our goal. Send in your application, if you have not already done so. Be the proud possessor of a membership in the Twelfth International Veterinary Congress. You will never regret it.

CAMPAIGN LAUNCHED AGAINST BANG'S DISEASE

The eradication of Bang's disease as a nationwide project is under way. The work has been started in three states—Maryland, Virginia and West Virginia. It is expected that Wisconsin, Minnesota, Oregon, Washington and the New England states will get started on the project shortly, the funds for which have been made available by the Agricultural Adjustment Administration, under the provisions of the LaFollette amendment to the Jones-Connally Act. The initial appropriation amounted to three million dollars and the work will be directed by the U. S. Bureau of

Animal Industry. The rules and regulations have been prescribed by Secretary of Agriculture Henry A. Wallace, as B. A. I. Order 347, which became effective July 19.

Under the plan the owners of cattle, which react to the agglutination test for Bang's disease and which are disposed of according to the regulations, will receive maximum federal indemnities amounting to $20 for grade animals and $50 for pure-breds. These are the same indemnities as those paid for reactors to the tuberculin test. With the exception of Virginia, none of the states are prepared to pay indemnities for Bang's disease at the present time. However, it is quite likely that several states will be asked to make appropriations for the purpose at the next legislative sessions in these states. Considerable work of a follow-up nature willl be necessary to render the preliminary work effective.

Present plans provide that the testing of Bang's disease be purely voluntary. This applies both to the states which participate in the work as well as to the individual cattle-owners within the several states. The first step on the part of the owner is to sign an agreement (1) to market for slaughter, under state or federal supervision, all heifers over six months old, cows and bulls that react to the agglutination test, (2) to confine additions to his herd, as far as practicable, to virgin animals and to those from herds known to be free of Bang's disease, (3) to continue blood-testing the animals in his herd in accordance with the accredited Bang's disease herd plan of his state, and (4) to clean and disinfect his premises under supervision after the removal of reactors. The use of abortion vaccines on any animals in the herd, during a six-month period prior to testing, is prohibited.

Dr. A. E. Wight, chief of the Tuberculosis Eradication Division of the U. S. Bureau of Animal Industry, is in charge of the Bang's disease project. Two million dollars for emergency tuberculin testing also has been made available and this work was started June 18.

SUFFICIENT REASON

A really professional and ethical veterinarian does not have to look very far or think very hard to find a reason why he should not sell anti-hog cholera serum and virus to farmers or so-called permit-holders. Neither is it difficult to find reasons why some veterinarians have been tempted to embark upon this rather ques-

tionable enterprise. As a matter of fact, the reasons are several. Hence, it becomes necessary for the veterinarian who is in doubt to weigh the facts in the case carefully, one against the other.

The vaccination of hogs has been "getting away" from veterinarians in certain sections of the country during recent years. Undoubtedly, the most powerful influence that has been responsible for this trend has been the county farm bureau. Then we have the overzealous, misguided county agent, in his quasi-official capacity, preaching the gospel of farmer vaccination. Aiding and abetting the county agent and farm bureau, we have the unethical serum companies who depend upon sales to farm bureaus, permit-holders, lay vaccinators and a few shortsighted veterinarians, in order to remain in business.

A comparatively recent move upon the part of the unethical serum companies was to place stocks of serum and virus in local drug stores, so that these products would be conveniently available to the trade. Nothing really new in this idea, after all. However, in these days of highly competitive merchandising, a move upon the part of one seller usually is met by a countermove upon the part of another seller in the same field. This is just exactly what happened in the serum industry. Somebody conceived the idea of using the local veterinarian for selling serum and virus to farmers. Briefly, that appeal went something like this: Doctor, why allow your clients to purchase their serum and virus from the farm bureau or drug store, and thereby crowd you out of the picture completely? Why not get into the business to the extent of supplying your clients with serum and virus, and take a profit on every order?

It is gratifying to know that the great majority of veterinarians have spurned these offers, if not for one reason, then for another. Good reasons are not hard to find. There is one, however, that is more important than all of the others put together. It is based on the principle that it is fundamentally unsound to place the living virus of an infectious disease in unskilled hands. The two most important veterinary organizations in the country —the American Veterinary Medical Association and the United State Live Stock Sanitary Association—are on record as being opposed to the use of hog cholera virus by laymen, believing such practice to be dangerous and not to the best interests of the live stock industry. Neither organization has ever receded an inch from the stand taken. Any veterinarian who sells virus to a layman repudiates the action of these two national organizations.

APPLICATIONS FOR MEMBERSHIP

(See July, 1934, JOURNAL)

FIRST LISTING

GLOSS, ELLIS H. Gaylord, Minn.
D. V. M., Iowa State College, 1931
Vouchers: R. A. Merrill and A. C. Spannaus.

HAGLER, CURTIS E. c/o John Morrell & Co., Topeka, Kan.
D. V. M., Colorado Agricultural College, 1932
Vouchers: C. B. Clement and N. L. Townsend.

HALL, FREDERICK A. 106 S. Cowen, Garrett, Ind.
D. V. M., Cornell University, 1923
Vouchers: C. R. Baumgartner and H. E. Bryan.

KILLIAN, GILBERT R. 768 E. Big Ben Blvd., Webster Groves, Mo.
D. V. M., Kansas State College, 1924
Vouchers: S. W. Haigler and H. S. Richards.

PARKER, G. THATCHER Box 14, Red Bank, N. J.
D. V. M., Cornell University, 1932
Vouchers: John B. Hopper and E. R. Cushing.

PAUL, S. G. Clarence, Iowa
D. V. M., Iowa State College, 1934
Vouchers: C. D. Lee and S. H. McNutt.

PHILIPSEN, W. H. Brandon, Vt.
B. V. Sc., Ontario Veterinary College, 1934
Vouchers: L. H. Adams and G. N. Welch.

SOMMER, HENRY L. 1210 Steiner St., San Francisco, Calif.
V. M. D., University of Pennsylvania, 1909
Vouchers: S. T. Michael and Maj. E. L. Nye.

STOVER, HENRY H. Langhorne, Pa.
V. M. D., University of Pennsylvania, 1934
Vouchers: F. E. Lentz and H. C. Terry.

WESTON, B. M. Asheboro, N. C.
D. V. M., United States College of Veterinary Surgeons, 1916
Vouchers: Wm. Moore and J. H. Brown.

Applications Pending

(See July, 1934, JOURNAL)

SECOND LISTING

Bader, Madero N., 1106 Avenue K, Galveston, Texas.
Clark, E. M., 4120 S. 20th St., Omaha, Nebr.
Danner, Howard, 1725 S. Meridian St., Indianapolis, Ind.
Elmer, Sam, New Glarus, Wis.
Franz, G. A., 4321 Emmet St., Omaha, Nebr.
Hamann, E. E., 2601 E. Michigan Ave., Lansing, Mich.
Jones, Eugene C., 9088 Santa Monica Blvd., West Hollywood, Calif.
Kermen, Wm. R., 932 Venice Blvd., Los Angeles, Calif.
Lang, Edward M., Jr., 716 E. Broadway, Louisville, Ky.
LiRocchi, Vincent W., 2820 S. Claiborne Ave., New Orleans, La.
Miller, Luther P., 114 E. Maple St., Clyde, Ohio.
Patterson, E. E., 3800 Grand River Ave., Detroit, Mich.
Patterson, Jas. E., 14411 Saint Marys Ave., Detroit, Mich.
Piercy, Paul L., University of Missouri, Columbia, Mo.
Winslade, W. A., Shipman, Ill.

The amount which should accompany an application filed this month is $7.08, which covers membership fee and dues to January 1, 1935, including subscription to the JOURNAL.

COMING VETERINARY MEETINGS

Saint Louis District Veterinary Medical Association. Melbourne Hotel, Saint Louis, Mo. August 1, 1934. Dr. Harley B. Wood, Secretary, 2754 Meramec St., Saint Louis, Mo.

Northwestern Ohio Veterinary Medical Association. Bowling Green, Ohio. August 2, 1934. Dr. W. P. S. Hall, Secretary, Division of Health, 9 Ontario St., Toledo, Ohio.

Poultry Science Association. A. & M. College of Texas, College Station, Texas. August 7-10, 1934. Prof. D. H. Reid, President, A. & M. College of Texas, College Station, Texas.

Tulsa County Veterinary Association. Tulsa, Okla. August 9, 1934. Dr. J. M. Higgins, Secretary, 3305 E. 11th St., Tulsa, Okla.

Interstate Veterinary Medical Association. Elks Building, Omaha, Nebr. August 13, 1934. Dr. G. L. Taylor, Secretary, Plattsmouth, Nebr.

San Diego County Veterinary Medical Association. San Diego, Calif. August 14, 1934. Dr. L. K. Knighton, Secretary, 3438 Mountain View, San Diego, Calif.

Twelfth International Veterinary Congress. Waldorf-Astoria Hotel, New York, N. Y. August 13-18, 1934. Dr. H. Preston Hoskins, General Secretary, 221 N. La Salle St., Chicago, Ill.

American Veterinary Medical Association. Waldorf-Astoria Hotel, New York, N. Y. August 14-16, 1934. Dr. H. Preston Hoskins, Secretary, 221 N. La Salle St., Chicago, Ill.

Southern California Veterinary Medical Association. Chamber of Commerce Building, Los Angeles, Calif. August 15, 1934. Dr. T. G. Beard, Secretary, 3684 Beverly Blvd., Los Angeles, Calif.

American Animal Hospital Association. Waldorf-Astoria Hotel, New York, N. Y. August 17, 1934. Dr. A. R. Theobald, Secretary, 4545 Reading Road, Cincinnati, Ohio.

Kansas City Veterinary Association. Baltimore Hotel, Kansas City, Mo. August 21, 1934. Dr. C. C. Foulk, Secretary, 1103 E. 47th St., Kansas City, Mo.

American Public Health Association. Pasadena, Calif. Sept. 3-6, 1934. Willimina Rayne Walsh, Secretary, 50 W. Fiftieth St., New York, N. Y.

CONTAGIOUS ECTHYMA (SORE MOUTH) OF SHEEP AND GOATS*

By I. B. BOUGHTON *and* W. T. HARDY
Texas Agricultural Experiment Station, Substation 14
Sonora, Texas

Contagious ecthyma is a specific contagio-infectious disease of sheep and goats, characterized by the formation of papules, vesicles, pustules and scabs on the skin of the lips. Swelling and inflammation of the infected lips accompanies the development of the lesions, preventing the infected lambs from suckling and grazing, as a result of which the affected animal loses weight and condition at a critical period of its life. The disease occurs principally in lambs and kids, but is often seen in a milder form in adult animals.

FIG. 1. Contagious ecthyma (sore mouth) in a kid.

In western Texas, the infestation of the swollen, inflamed lips with the larvae of the screw-worm fly (*Cochliomyia macellaria*) constitutes the most serious aspect of the disease and annually causes lamb and kid losses amounting to hundreds of thousands of dollars. The disease has been prevalent in the Edwards Plateau and Trans-Pecos regions of this state for at least 25 years, especially since the fencing of the range and the consequent

*Presented at the seventieth annual meeting of the American Veterinary Medical Association, Chicago, Ill., August 14-18, 1933.

"looseherd" method of ranching. It should be borne in mind that the sheep population of this region totals in excess of 4,000,000 sheep, while reliable estimates fix the Angora goat population at approximately 2,000,000 head.

Review of the literature shows that the disease occurs in many parts of the world and has been studied by several investigators. Zeller,[1] in 1920, published his findings as a result of his work with lip scab forwarded to him in Berlin by a veterinarian who had secured the material from goats in the province of Otjiwarango in Southwest Africa. Zeller was successful in transmitting the disease to both sheep and goats with this material.

FIG. 2. Screw-worm damage on lip of kid, following sore-mouth infection.

In 1923, Aynaud[2] reported on "chancre du mouton," identifying it as the same disease as Moussu's "ecthyma contagieux des levres," and described a severe form which invades the mouth and a benign form which remains confined to the lips. This author discusses the disease rather fully, stating that it has a wide distribution and is of frequent occurrence in France, especially since the war.

Moussu[3] claims that the name "contagious pustular stomatitis," as used by Aynaud, was wrong, inasmuch as the lesions may remain strictly localized on the lips. Blanc, Melanidi and Caminopetros,[4] in 1922, published on the same condition in goats in an

article entitled, "Recherches experimentales sur une maladie eruptive de la chevre, observée en Grèce." They were successful in transferring the disease by inoculation of the emulsified crusts into the scarified skin of the thigh, following the procedure of Aynaud.

Jocotot,[5] in 1924, reported a similar condition among goats in Annam, Indo-China, stating that these animals were especially susceptible. He noted that the submaxillary glands are inflamed and that a durable immunity follows recovery. Lanfranchi[6] reported the existence of a similar disease in sheep in Italy. They note the establishment of immunity following both the natural and the experimental disease, stating that such immunity is not always lasting and may disappear five to eight months subsequent to infection.

Theiler,[7] in 1928, in South Africa, published a rather complete description of both the natural and the experimentally-induced disease, as it occurs in South Africa, under the name of ecthyma contagiosum. He succeeded in transmitting the disease with material from the lip lesions of infected sheep but was not successful in filtering the virus, stating, however, that in view of the difficulty of successful filtration as reported by Aynaud, Zeller and others, he did not consider such failure as conclusive proof that the causative agent was not a filter-passer. Aynaud, with diluted fresh lymph, filtered the virus through earthenware candles but states that positive results are difficult to obtain. According to Theiler, Zeller succeeded in only one-third of his filtration experiments with Berkefeld V and Nordtmeyer Berkefeld filters. The same difficulty in filtering the virus is reported by every investigator who has worked with the disease. Further, Theiler discusses the nature and resistance of the virus and mentions the possibility of vaccination, referring to the work of Aynaud in France. He emphasizes the fact that the outbreaks he has observed were all benign in character and that there were no secondary complications.

Melanidi and Stylianopoulo,[8] in 1928, reported the disease as occurring for the first time in Greece despite the earlier report of Blanc and associates. Melanidi and Stylianopoulo reported mortalities varying from 5 to 60 per cent and stated that the disease was identical with that reported from France. According to these authors, the high mortality was due to secondary involvement of the lungs and intestinal tract; they observed typical lesions on the lips and in the mouth and record swelling of the regional lymph-glands accompanied by fever. In their hands in-

tracutaneous vaccination with scab emulsion protected susceptible animals.

Glover[9] reported on an eruptive disease of the lips of sheep in England, stating that it was evidently analogous to the disease reported from the Continent. He found that sheep and goats alone are susceptible and that the virus is filtrable only with difficulty. This author calls the condition contagious pustular dermatitis. Further, Glover succeeded in transmitting the disease with the dry scab in a dilution of 1:50,000, and remarks on the resistance of the virus to dessication and noted immunity resulting after an attack of the disease. This investigator was not able to transmit the infection to susceptible animals using the defibrinated blood, drawn from infected animals at the height of the disease, as the inoculum. Similarly, he failed to produce skin lesions either by the intravenous or the subcutaneous inoculation of the virus into susceptible animals, a finding confirmed by the experiments of the present authors. This author stated, however, that such inoculations did confer some degree of immunity. Lastly, Glover, as the result of several experiments, agrees with other investigators that inoculation of susceptible animals with properly prepared material from the lip lesions induces the formation of a benign local lesion which sets up a definite, solid immunity that endures for at least eight months.

In 1929, Schmidt and Tunnicliff,[10] in Texas, reported successful transmission of the disease to susceptible animals, mentioning the fact that they were investigating a practical method of vaccination. These authors further noted that the disease is prevalent in western Texas, under the name of sore mouth.

Howarth,[11] in 1929, published on outbreaks of the disease under the name of infectious pustular dermatitis, in two separate flocks of sheep in California. He had no difficulty in transmitting the disease through infectious material from natural cases. This author was unable to find *Actinomyces necrophorus* in the lip lesions but did isolate *Staphylococcus aureus* in pure culture. Like all other investigators, Howarth reports difficulty in filtering the virus, stating that uniform results could not be obtained with each filtration. He noted that one attack conferred a high degree of immunity and that infected animals, if left untreated, soon recover.

Jungherr and Hardy,[12] in 1930, reported several experiments with lambs and kids at the Texas Station. They demonstrated that the disease is readily transmissible through contact and found that it is transmitted naturally from kid to kid but only rarely from kid to lamb.

Newsom and Cross,[13] in Colorado, reported complications with *A. necrophorus* among feed-lot lambs infected with sore mouth. These authors describe lesions developing about the mouth and gums, and in the pharynx and esophagus, with occasional involvement of the membrane of the reticulum, leaves of the omasum, intestinal mucosa and the lungs. They were able to demonstrate this organism in such lesions.

Schmidt and Hardy[14] refer to a report by Willems[15] that the disease occurs in Belgium and suggests the possibility of its transmission to men. Hatziolos[16] reported that occurrence of contagious ecthyma among sheep in Greece, stating that goats running with the infected sheep did not contract the disease. He observed that the disease is evidently transmissible to man since he noted typical lesions on the lips, gums, tongues and cheeks of shepherds handling infected animals. This author vaccinated susceptible sheep with good results.

Schmidt,[17] in 1931, reported the successful vaccination of young lambs and kids. He further remarked that the age of the animal seems to influence its resistance to the virus of the disease. Seddon and Belschner[18] described the same disease from Australia, where it is commonly known as scabby mouth, in 1930, noting immunity from one attack of the disease and stating that susceptible animals could be immunized by inoculation with scab emulsion, the technic of such inoculation being analogous to that of smallpox vaccination in the human subject. He describes lesions on the skin of the face (around the nostrils, eyes and ears), of the coronet and the udder as well as the mucous membrane of the lips, gums, palate, dental pad and tongue. This author states that infected lambs sometimes transmit the disease to the skin of the udder of a susceptible ewe, the induced lesions being typical of the lip form of the disease. In a later report, Seddon and McGrath[19] demonstrated that English and Australian strains of the virus, when inoculated into susceptible animals, confer a solid immunity against each other as well as against homologous strains.

Schmidt and Hardy[14] gave a very good description of the disease as it occurs in western Texas. They note the negligible mortality of the disease itself but emphasize the economic importance of the loss of weight by infected lambs resulting from the deformation of the lips. Further, these authors discuss the large loss of infected lambs as a result of the injurious activities of the *C. macellaria* larvae.

They were not successful in obtaining positive filtrates of scab emulsions, in six different experiments. In these tests Seitz

asbestos filters were used in three and Mandler diatomaceous filters in the other three. Further, these writers note that a solid immunity develops following a natural or an induced attack of the disease, regardless of the location of the skin lesion and that such lesion remains localized. Charts of immunity tests are given, showing that animals which have been vaccinated are immune to subsequent exposure to the virus.

ETIOLOGY

The results of our filtrations of emulsified scab taken from natural or vaccination cases of sore mouth have not always been uniform, but in several instances we have secured positive filtrates, using Mandler filters. The technic of the filtration was the same in every case. The filters were new, unused, freshly washed in distilled water and dried in the hot-air oven, wrapped and then sterilized in the autoclave for 20 minutes at 20 pounds pressure. Before filtration, the entire apparatus used for filtering (candle, candle-tube, filtering flask, and rubber tubing) was assembled wrapped completely in paper and autoclaved for 20 minutes at 20 pounds pressure.

The material to be filtered was an emulsion of the dry scab in sterile normal saline or distilled water in a dilution of 1:100, except in one test. In this case, hormone broth, made as described by Tang[20] except that mutton heart was substituted for beef heart, was used as the suspending vehicle, with positive results as regards the infectiousness of the filtrate. In three cases, two of which were successful, the emulsion was centrifuged at 2,000 R. P. M. for 15 minutes, the supernatant fluid poured off and filtered, the residue in the bottom of the centrifuge-tube being used for the inoculation of the control animals. In the three other tests, two of which gave positive filtrates, the scabs were ground in a sterile mortar, emulsified in the diluent by hand shaking and then poured into the candle-tube.

The emulsion was poured into the candle-tube and the filtrate sucked through the candle by a vacuum pump. In no case did the filtration take longer than 20 minutes. In all cases the filtrate was cultured, aerobically and anaerobically, using both solid and liquid media, and incubated at 37.2° C. for six days. At the end of this time, no growth having developed in either the aerobic or anaerobic tubes, the cultures were discarded.

Table I shows the results of one such filtration. It will be seen that one susceptible lamb (428) developed a very mild case of sore mouth while the other (429) showed a severe, positive reac-

TABLE I—*Sore mouth transmission test with sterile filtrate of potent scab, June 14 to July 14, 1932.*

LAMB	AGE (MOS.)	TREATMENT JUNE 14	OBSERVATIONS		RESULT
			JUNE 20	JUNE 26	
428	6	Inoculated on scarified skin of belly and lips with 1 cc each of a sterile filtrate of sheep contagious ecthyma scab*	Four pustules on belly, surrounded by intense inflammation. Lips: a few pustules surrounded by intense inflammation	Lips: healed. Belly: dry scab removed. Healed underneath. Released	Sore mouth +. Healed on June 26, 1932
429	6	Inoculated on scarified skin of belly and lips with 1 cc each of a sterile filtrate of sheep contagious ecthyma scab	Sore mouth ++++. Pustules on both lips and belly. Intense inflammation	Inflammation, scabs and pustules on belly. Lips: upper left, swollen and inflamed, scab formation; lower left, swollen, inflamed scab formation. Numerous pustules present. Photographed	Sore mouth ++++. Healed on July 14, 1932
427 Control	4	Inoculated on scarified skin of belly with 1 cc of residue in candle-tube after filtration of the scab	Inflammation, pustules and some moist scab on scarified area of skin of belly	Inflammation, swelling and moist scab on scarified area of belly	Sore mouth ++++. Healed on July 8, 1932

*These scabs were diluted 1:100 with sterile distilled water and filtered through a Mandler N filter. Cultures of this filtrate remained sterile after six days.

tion which healed after 30 days. The control animal (427) developed a typical case of the disease, the inoculation site being clean after 24 days. The difference in the reactions of lambs 428 and 429, to the same virus, may have resulted from a difference in susceptibility since it has been found that in very susceptible lambs, an average interval of about 25 days is required for complete healing of the experimental disease. On the other hand, the mild reaction in lamb 428 may have been due to the fact that only a very minute quantity of the virus was contained in the drop of filtrate which was rubbed into the scarifications.

Altogether, we have made six filtration tests, all with Mandler filters, and have secured four positive filtrates. These results confirm the findings of other investigators that the filtration of the virus is carried out with more or less difficulty and that uniform results are not always obtained.

We have not been able to find *A. necrophorus* in the lesions of either field or experimental cases of sore mouth. In searching for this organism, we inoculated one pair of rabbits subcutaneously with a scab emulsion. These animals remained healthy. One of them was killed and autopsied approximately one month after injection, no lesions of *A. necrophorus* infection being found. We could not demonstrate this organism in smears from the liver, and all anaerobic cultures remained sterile. Repeated anaerobic cultures (using the pyrogallic acid method) of the pustular fluid and of scab emulsions have been consistently negative for any growth, and we have never been able to demonstrate this organism in smears made from typical lesions. Inasmuch as we have never encountered any cases of sore mouth, whether natural or experimentally induced, which failed to heal normally if protected from *C. macellaria* but untreated otherwise, and which left the site of the lesions smooth and without scars, we are forced to conclude that the necrophorus organism evidently plays no part in the disease as it occurs in this region.

Repeated culturings of the pustular fluid of typical lip lesions of both natural and inoculation sore mouth have shown *S. aureus* in pure culture in every instance. In securing the material for culture the thin skin covering the pustule was removed aseptically with sterile forceps, a sterile inoculating needle thrust into the pustule, removed and the material transferred immediately to the tubed medium. This organism seems to be present constantly in the lesions of the disease in this region as it does in the cases reported by Howarth,[11] and Seddon and Belschner.[18]

Strains of *S. aureus*, isolated from typical cases and grown on nutrient agar in the laboratory, have been inoculated into the

lightly scarified lips of susceptible lambs in several instances. At the site of inoculation there is slight inflammation after 24 hours and the formation of small pimples but the animal evidences no pain in eating or suckling. The pustules dry up, with only a very slight flaky scab being formed, the inflammation subsides and the lips present a normal appearance within four or five days after the inoculation. Such findings definitely eliminate this organism as a causative factor in view of the fact that the inoculated lip, when lightly scarified and rubbed with the virus of contagious ecthyma, is just beginning the pustule formation about the fifth or sixth day. Seddon and Belschner[18] state that only in cases of deep puncture of the skin with an infected needle can *S. aureus* produce a pustular dermatitis and that such dermatitis is not typical of sore mouth.

The relation of lip-and-leg ulceration of sheep, as described by Melvin and Mohler,[21] to contagious ecthyma has not been worked out definitely but the similarity of lip lesions in the two conditions indicates the probability that the former condition is originally caused by the virus of contagious ecthyma and that *A. necrophorus* enters the picture as a secondary invader. While the lip lesions described by these authors resemble very closely those of the disease under discussion, we have never observed the coronary ulcers although many of the sheepmen in this region state that such lesions are often present on badly affectd animals. The fact that the virus of contagious ecthyma can set up the typical lesions when inoculated into the skin of any part of the body strengthens the view that leg (coronary) lesions may very well be present in some cases, the infection being transferred from the infected, swollen lips to the coronet by the animal itself. Obviously, there would be more chance for such lesions to become infected with *A. necrophorus* than there would be for similar infection of the lip lesions.

So far, neither the writers, nor any of the investigators reporting on this disease, have found any indication of plurality of virus among the same species of animals. It is worthy of note that goats, running in the same pastures with badly infected sheep, many times do not contract the disease, while we have reports from ranchmen that they have sore mouth every year among their goats, but sheep in the same pasture remain free from the infection.

The virus contained in the scabs is very resistant, remaining potent for at least 15 months (at this laboratory) when kept at 14° C. in brown glass bottles. Aynaud,[2] Seddon and Belschner,[18] and Theiler[7] record the presence of virus in dry scabs after more

than a year. While pure glycerin seems to have a slow killing action on the virus, the scabs, emulsified in equal parts of normal saline solution and glycerin, retain their virulence for at least one year when kept in the cooler at 14° C. While no definite reports are available as to the length of time scabs on the range retain their virulence, the fact that the disease often reappears, after an absence of two or three years from a particular range, indicates that the virus is extremely resistant to climatic conditions. We have found that the virus in the scabs may be killed, somewhere between 30 and 60 days, when exposed on the ground to weather conditions during the summer, which is usually very dry and hot in this region. The fact that one lot of scabs was so killed does not indicate, however, that the same fate attends all the scabs dropped on the range. Scabs, exposed to the weather on the ground, during the fall and winter months, retained their virulence when tested in the spring. This result proved that the virus will carry over easily from the early fall until the spring, when a new crop of lambs is dropped.

Contagious ecthyma belongs to the group of eruptive, infectious diseases of the variola-vaccinia group but that the disease is distinct from sheep-pox is evident. In contagious ecthyma there is no generalized eruption over the hairless parts of the body as in sheep-pox, nor have we been able to demonstrate any systemic disturbance in the inoculated animal subsequent to inoculation. In natural outbreaks, the lesions of contagious ecthyma are confined to the lips and the udder, indicating that the infection is a localized affair resulting from direct infection of the affected area. There is little question that the udder lesions in ewes are the result of direct infection by the swollen, inflamed lips of the lamb during suckling. In inoculation into the scarified skin, we find that the lesions develop only at the site of inoculation and do not spread. As reported by Glover[9] and confirmed by the writers' experiments, intravenous and subcutaneous inoculation of susceptible animals with the virus of contagious ecthyma fail to produce lesions of any sort, nor are any general systemic symptoms manifested.

Cross-immunity tests, carried out by the Greek workers, Blanc, Melanidi and Caminopetros,[4] using both contagious ecthyma virus and sheep-pox virus, demonstrated that it was not possible to immunize lambs against sheep-pox with contagious ecthyma virus and vice versa.

Contagious ecthyma virus can be strongly diluted and still retain its infectivity. Glover[9] and Howarth[11] reported "takes" with

virus in 1:50,000 dilution, while the writers have transmitted the infection to a susceptible kid with scab material diluted 1:100,000 with sterile normal saline.

The thermal death point of this virus, which we found to be 58-59° C., is in the same range as the other viruses of the variola group. Aynaud[2] reported the thermal death point as 56° C. after heating for 30 minutes.

In table II we see the results of the inoculation of susceptible animals with sterile positive filtrate which had been heated in a water-bath at 58-59° C. for 30 minutes. It will be noted that the control animal inoculated with the unheated virus contracted the disease but that the animals inoculated with the heated virus showed no evidence of the disease. These three lambs (428, 429 and 430) later were inoculated with known virulent virus, all three of them developing typical lesions of contagious ecthyma. Virus heated in the water-bath at 55° C. for 20 minutes still retained its virulence when inoculated into the skin of susceptible lambs.

In accord with other investigators, the writers have been able to transmit the infection only to sheep and goats. Inoculation of infective virus into the scarified skin of the rabbit, guinea pig, a calf and a dog in controlled experiments gave negative results for the tested animal species and positive results in the case of the controls. Instillation of known potent filtrate into the cornea of rabbits likewise proved negative. Aynaud[2] reported successful transmission of the disease to calves through intracutaneous inoculation.

Observations, both in natural outbreaks in the field and in inoculated cases in controlled experiments, show that young animals (lambs and kids) are extremely susceptible and suffer most from the disease. There is little question that resistance to the disease increases to some extent with age but tests, both by the writers and by Schmidt and Hardy,[14] demonstrate that aged sheep and goats remain susceptible if they have not undergone a previous attack of the disease. Observation has shown definitely, however, that older animals seldom, if ever, show the severe type which is the typical form encountered in lambs and kids. Among yearlings and older sheep mild lesions of the disease often are seen on the lips and occasionally on the udder but we have observed only a very few cases of the severe type in animals over one year of age. Some ranchmen have reported severe sore mouth in long-yearling sheep but we have had no opportunity of observing such cases at first hand.

TABLE II—*Potency test of sterile filtrate of sheep contagious ecthyma scab heated in a water bath at 60° C. for 30 minutes, May 17 to May 23, 1933.*

LAMB	AGE (WKS.)	TREATMENT MAY 17	OBSERVATION MAY 23	RESULT
428	4	Inoculated on scarified skin of belly with 1 cc of sterile filtrate 388 of sheep contagious ecthyma scab heated at 60° C. for 30 minutes	Dry scratch scab. Healed. Released	Negative for sore mouth
429	4	Inoculated on scarified skin of belly with 1 cc of sterile filtrate 388 of sheep contagious ecthyma scab heated at 60° C. for 30 minutes	Dry scratch scab. Healed. Released	Negative for sore mouth
430	4	Inoculated on scarified skin of belly with 1 cc of sterile filtrate 388 of sheep contagious ecthyma scab heated at 60° C. for 30 minutes	Dry scratch scab. Healed. Released	Negative for sore mouth
397 Control	3	Inoculated on scarified skin of belly with 1 cc of sterile filtrate 388 of sheep contagious ecthyma scab	Pustules. Intense inflammation. Sore mouth ++++	Sore mouth ++++
398 Control	2	Inoculated on scarified skin of belly with 1 cc of sterile filtrate 388 of sheep contagious ecthyma scab	Pustules. Intense inflammation. Sore mouth ++++	Sore mouth ++++

The disease appears to be transmitted, in a mild form, to man, as suggested by Willems[15] and Hatziolos.[16] Lesions clinically identical with those of contagious ecthyma in sheep and goats sometimes are found on the hands of men handling infected animals during the annual outbreaks of the disease. One of the writers (W. T. H.) developed a rather typical pustule on the skin of the right wrist a few days after handling infectious material. Inoculation of material from this lesion into the scarified skin of the thigh of a susceptible lamb was followed by the development of typical sore mouth lesions which reached the pustule stage only and then dried up without forming a scab.

Schmidt and Hardy[14] discuss the apparent resistance of sheep to the goat virus, stating that they observed lesions of a mild type when the goat virus was inoculated into the lightly scarified skin of susceptible sheep. The present authors, in working with sheep virus, succeeded in giving typical, severe lip lesions to goats through inoculation of the scarified skin with potent sheep virus. The question as to whether there is a sheep strain and a goat strain of the virus needs to be studied further. Vaccination of goats with sheep virus will protect against subsequent exposure to sheep virus after the vaccination scab has dropped and the site of inoculation is clean but we have had, thus far, no opportunity to test the vaccination of sheep with pure goat virus. Arguing by analogy, we believe that it will immunize sheep against sheep virus but we have no experimental data to back up such a statement. If sheep, inoculated with goat virus, develop a mild type of lesion but build up enough immunity to protect against field exposure to the disease, it is probable that a vaccine made from goat virus will be much more practical than one made from a sheep strain.

GEOGRAPHICAL DISTRIBUTION

The distribution of the disease in the United States is not definitely known. Contagious ecthyma is prevalent throughout the sheep and goat country of western Texas. The writers, during the past winter, working with lip scab supplied by Dr. T. O. Brandenberg, state veterinarian of North Dakota, and Dr. R. E. Shigley, of the same state, proved that the disease also exists in North Dakota. Dr. H. Schmidt, of the Texas Agricultural Experiment Station, in a personal communication, stated that he had reproduced typical sore mouth in susceptible lambs with lip scab supplied from Colorado lambs by Dr. I. E. Newsom, of that state. Reports from feeders in Morris, Kansas, state that a type of sore mouth, clinically identical with the disease under dis-

cussion, appears among feeder lambs usually within ten days after such animals are put in the feed-lots. From descriptions of more or less similar conditions occurring in sheep, as reported in various veterinary journals from time to time, the writers are convinced that this disease is widely distributed throughout the sheep-raising parts of the United States. Further, the disease is reported as prevalent among sheep in the northern provinces of Mexico.

From the literature, we find that it occurs in Australia, New Zealand, South Africa, Morocco, France, Greece and England. According to Seddon and McGrath,[19] the distribution of the disease is world wide.

Occurrence in Western Texas

The natural disease as it occurs on the range does not appear sporadically in a band of lambs or kids but seems to break out in a number of them either at the same time or in very close succession. The ordinary report of the ranchman is that he finds a few cases today and several more on successive days until, in many cases all the lambs in the band are infected. As mentioned above, it is the lambs or kids which are severely affected in the natural outbreaks but several light cases are usually noted among the older animals. The annual outbreaks in this region appear during the early spring months, April and May, in some sections; a little farther north the infection usually appears during June and July, while a still more northerly section reports the disease during August and September. But for all practical purposes, the disease is prevalent in western Texas during the hot summer months, subsiding with the approach of cooler weather in the fall. Occasionally, the disease appears in a mild form among short-yearling lambs in the winter months along the Rio Grande border.

Natural Disease

The first symptoms noted on the range are usually the more or less warty, moist scabs and the swollen lips; this swelling gives the face of a lamb a peculiar appearance, which can be noticed from some distance away. These scabs are usually a brownish gray in color and moist in the early stages. Later they dry out, becoming hard and very often cracked. When removed manually or torn off by brush or weeds, they leave an elevated, raw, bleeding surface, which attracts the screw-worm flies. If not infested with screw-worm larvae, the scabs gradually dry up, finally loosening and dropping away, leaving the skin of the lips smooth and without scars.

Close observation may show the beginning of the process in natural outbreaks but the development of the lesions is best studied in inoculated animals. In range outbreaks, if closely examined, we find that the papules, followed by vesicle, pustule and scab formation, accompanied by inflammation and swelling, are usually confined to the skin of the lips but are sometimes scattered over the skin of the nostrils and around the eyes. Aynaud[2] and other investigators describe the presence of the typical lesions on the mucous membrane of the mouth, pharynx, dental pad, and even on the digestive mucosa, but we have observed only a relatively few cases of buccal mucous membrane lesions among thousands of infected animals examined. Apparently, the lesions in this region are confined almost altogether to the skin. It is noted in the development of the lesion that the small vesicles usually are single, there being little tendency for them to coalesce; they may be very numerous and very close together but it is not until the pustules, developing from the vesicles, rupture and discharge their contents, that they coalesce and form a more or less compact scab. In many mild cases the scabs remain separate.

After a day or so, the vesicle develops into a pustule which is filled with a yellowish-white fluid material and covered with the thin, white, glistening epidermis. Twenty-four to 48 hours later, the thin skin ruptures and the ulcerated surface begins to scab over. During the development of this ulcer, the lips become swollen, sometimes enormously, are intensely sensitive and very stiff. The lamb so affected cannot suckle the ewe nor can it graze to any extent. It presents a miserable appearance and loses weight rapidly.

In our observations we have not found that even badly infected lambs show either fever or inappetance during any stage of the disease. Their attempts to suckle and to graze are almost entirely defeated by the severe swelling and sensitiveness of the lips.

Scab formation takes place gradually over the raw surface. The attack from the initial lesion, until the scab has dropped, averages approximately 25 days, providing the healing process is not retarded or stopped completely by invasion of the lesions with the larvae of *C. macellaria*. We have not seen any secondary complications of microbic nature which interfere with the evolution of the disease process nor which cause any mortality among the lambs. As stated above, the mortality from contagious ecthyma *per se* is negligible in this area, but the loss in weight is of primary importance to the sheep-owner.

In many different outbreaks and even in the same flock, we have observed much milder types of lesions in some animals than in others. Individual animals may show merely a light scab formation on the lips with no marked swelling and little, if any, evidence of pain. The scabs may remain separate and distinct, each scab evidently developing from a single vesicle. But the characteristic raw, elevated, bleeding surface usually is seen under all scabs if they have been removed before they have progressed far enough to loosen in the natural way.

Experimental Disease

The development of the lesions can be studied best in the inoculated animal. In all our inoculation experiments we have followed the same technic. The skin, sometimes of the lip and sometimes of the flank, was lightly scarified and the scab emulsion or the filtered virus was rubbed in thoroughly. The scarifications were made deeply enough to break through the superficial layer of the skin into the dermis but not sufficiently deep to bring the blood.

Forty-eight to 72 hours subsequent to inoculation, a zone of inflammation can be seen surrounding the scarified area. In some cases this inflammation appears in small circles, apparently surrounding a small area where the virus was deposited, but in the majority of cases the entire scarified area is enveloped by the inflammation. Within 24 hours after the appearance of the inflammation, close examination reveals the presence of many small papules, circular in shape and resembling those of all other pox-like diseases. Within another 24-hour period, small vesicles, filled with a clear fluid, are noted protruding from the papules. Concomitantly with this are noticed a swelling of the skin of the scarified area and an increase in the intensity of the inflammation.

Usually by the next day it can be noted that the vesicles have become pustular in character, with the contained fluid of a whitish-yellow color and increased in quantity. At this time the epidermal covering is very thin and more or less tensely drawn, depending upon the quantity of fluid in the pustule. It is a common occurrence for the pustules to develop some little distance away from the scarified area, whether by extension through the tissue or by direct contact, we do not know. Experiments have shown, however, that the virus in some cases is capable of setting up the typical lesions when dropped onto the apparently unbroken skin.

All pustules do not form and rupture at the same time. We have noted pustule formation up to the twelfth day following inoculation, but in most cases pustules are not formed subsequent to the ninth day. About the sixth day the pustules rupture and discharge the contained fluid, the resultant lesion appearing very much like an ordinary ulcer. At this time the entire scarified area appears swollen, raw and intensely inflamed. The rupture of the pustules exposes the vital layer of the skin, and, in our experience, infection with pyogenic organisms, usually *A. aureus*, occurs at this time in many cases.

In the great majority of cases, however, the fluid from the pustule, together with the scavenger leucocytes and wound serum, gradually dries up and forms a scab. There is little question that the pustular fluid spreads the infection, to some extent, as attested by subsequent pustule formation more or less adjacent to the original pustule. The thickness of the scab is increased by the piling up from underneath of the leucocytes which have been concentrated here in response to the injury by the virus. Finally the scab is additionally thickened by the profuse proliferation of epithelial cells from the *rete Malpighii*. These scabs usually form as a homogeneous mass, attaining a thickness of one-fourth to one-half inch.

If the scab is removed manually at this stage, the raw, elevated, bleeding surface of the dermis is exposed. A peculiar, sweetish odor is very often noted emanating from the lamb during the stage of scab formation. If undisturbed, we note that the scab assumes a brownish color which gradually acquires a grayish tint. At this time, pus may often be expressed from underneath the scab by pressure. As a rule and particularly in the case of very thick scabs, fissures appear in several places. Very often, blood seeping through these fissures attracts the blow-flies which deposit their eggs, the subsequent development of the very active larvae interfering with the healing and too often leading to the death of the animal. Normally, the scab gradually dries up, drops off 15 to 20 days after scab formation has started and leaves the area smooth and without scars other than the marks of the scarification. In some cases, the dry scab gradually crumbles away, while in others it drops off as a single entity. The whole process of the disease occupies approximately 25 days. Obviously, there is considerable variation in the time, some of the milder cases healing completely within two weeks, while in the severe type, the scab may persist for as long as 40 days after the appearance of the initial lesion.

Occasionally, in inoculated animals, no distinct vesicular and

pustular formation is noted, the line of the scarification merely becoming surrounded by intense inflammation, elevated above the healthy skin and finally covered with the characteristic scab. Schmidt and Hardy[14] note the formation of hard knots in the skin immediately under the pustules, which gradually disappear within the course of a few weeks.

We have had little opportunity to examine the buccal mucous membrane lesions since they are of comparatively rare occurrence in this region. There is naturally no scab formation accompanying this type of lesion since the pustular exudate together with the scavenger leucocytes and the wound serum are continually swept away by the salivary secretions. The lesion appears as a soft, whitish-yellow spot, surrounded by a small area of intense inflammation.

Natural Infection

The fact that we have been able to transmit the disease with the fluid from typical pustules and with watery and saline emulsions of the lip scab (strongly diluted) demonstrated the means by which the infection is spread. In the natural disease the lesions are confined largely to the lips of the lamb. While the probability is that some slight irritation or scarification of the skin of the lips favors the entrance of the virus and the subsequent establishment of the disease, the fact that we have observed the disease following the application of the virus to the unbroken skin, shows that skin injury is not always essential for the entrance of the virus. It is obvious, of course, that the young animal has abundant opportunity to sustain slight scratches on the lips incident to grazing, particularly in this region where much of the range is dotted with woody brush of various kinds.

There can be little question that the original infection in an outbreak is picked up from virulent scabs on the range, but the rapidity with which the disease spreads in a flock argues against the probability that all subsequent cases are contracted in the same manner. The fact that extremely minute quantities of virus are capable of instituting the disease indicates that contamination of the water supply may furnish a common source of infection. In the opinion of the writers, direct contact between healthy and infected lambs is responsible, to a large extent, for the rapid spread of the disease through a flock in a pasture.

Economic Importance

The annual losses sustained by the sheep-owners of western Texas as a result of contagious ecthyma amount literally to hundreds of thousands of dollars every year. As stated previously,

the direct loss of life among lambs and kids from the disease *per se* is negligible, but the shrinkage in weight, resulting from an attack and occurring at a time when the young lamb should be growing and developing, occasions a tremendous loss. The most important loss, however, is due to the invasion of the infected lips by the larvae of the blow-fly, which is prevalent throughout the spring, summer and autumn months in this region. Thus far, no dependable, practical repellant for these flies has been found. As a consequence, lambs infected with sore mouth must be treated at least every other day to protect them from the attacks of these flies.

Some of the ranches have large, screened, fly-proof sheds where the infected animals are housed but, in the usual extensive outbreaks, such sheds are not sufficiently large to accommodate all the affected animals. In addition, under our ranching conditions, where the sheep are run in pastures which vary in size from one to five or more sections of rough country, often very brushy, the practicability of rounding up the animals every few days is extremely questionable. In many cases, the outbreak has secured a solid foothold and the great majority of the lambs may be infected before the rancher is aware of the fact. Since the disease usually appears at about the same time every year, the habit of the rancher is to ride the pasture every few days during the sore mouth season and to inspect the animals closely.

Infection of all the lambs in certain pastures, sometimes of all the lambs on a ranch, is not at all uncommon. As stated previously, the disease appears first in a few lambs and very quickly spreads (within one to two weeks) through the band. As a result of this, removal of infected lambs, together with their mothers, from the pasture to a place of isolation, is not practical, especially in view of the fact that the entire premises are probably infected with the virus of the disease. In addition it is impossible to gather any number of lambs in the pasture and also be sure that their ewes have also been gathered with them. This is of first importance, since it must be borne in mind that the lambs are still suckling their mothers and have not yet learned to graze to any extent.

If the infected lamb, with its raw, swollen lips infested with the blow-fly larvae, could be housed away from further attacks by these flies, it would be a simple matter to kill the larvae already present and allow the condition to heal naturally. But, due largely to the ranching conditions obtaining in this region, such a procedure is impractical to say the least. Hence the infected lamb is exposed to repeated infestations with the larvae

and the condition becomes daily more aggravated with new generations of larvae hatching out almost continuously.

Consequently, the mortality, resulting from the larvae injury, often reaches large totals, losses of 100 to 500 lambs on a ranch not being at all uncommon. While there is no exact way of estimating the damage to the infected lamb which recovers, there is little question that the loss resulting from this, combined with the loss due to the blow-fly damage, represents a tremendous financial loss to the sheep-owners of this entire region. One representative ranchman, who runs approximately 11,000 breeding ewes, estimates his annual loss from sore mouth at approximately 75 cents per lamb.

TREATMENT

Medical treatment of any kind, in our hands and as practiced by the ranchers, is unsatisfactory, to say the least. The virus, by the time the condition is noted on the range, is already within the skin and cannot be reached by the external application of medicaments. The use of pine-tar oil, for its germicidal effect, combined with some bland oil which tends to soften the scabs and protects to some extent against the blow-fly depositing its eggs, has given the best results. But this treatment must be repeated almost daily and at best is of little avail against the condition. There can be little question that the rancher many times spreads the disease among his lambs as a result of this method of treatment, handling, as he does, infected lambs and then treating the healthy lambs in a mistaken idea that he can thus prevent the occurrence of the disease in the latter animals.

IMMUNIZATION

The writers, in a series of immunization experiments, found that a vaccine made from the scabs secured from animals suffering from either the natural or experimental disease, consistently produced good "takes" when rubbed into the lightly scarified skin of susceptible lambs. The technic of the inoculation was simple, consisting of light scarification of the skin with a knife-blade or the point of a hypodermic needle, followed by the application of a small quantity (one drop) of the vaccine. Usually this was rubbed into the scarified area with a small stiff brush but many times the vaccine, when merely dropped onto a scarified area, induced good "takes."

In many experiments made to test the degree of immunity conferred by vaccination, the writers' results confirm, to a large extent, the findings of other investigators. That the vaccinated

animal is immunized against exposure to the disease, as it occurs on the range, is demonstrated by the results of the field experiments discussed in the following pages. In some cases, however, we find that the vaccinated animal is not completely immune to artificial reinfection when the virus is rubbed into the scarified skin. In some experiments designed to test this reaction, we found that the disease, upon reinoculation with known virulent virus, progressed to the pustular state only. The pustules dried up and a very thin, flaky scab formed, which dropped away within eight or nine days after the inoculation. Practically no swelling and no signs of sensitiveness were exhibited by the animal. Not all animals in these experiments reacted in this way, some of them showing no sign whatsoever of a "take," indicating that a solid immunity had been established. The foregoing experiments were conducted approximately one month after the scab from the original vaccination had dropped.

The fact that pustules are observed to form several days subsequent to the rupture of the first pustule indicates that the establishment of the immunity is relatively slow. Observations have shown that the immunity is not thoroughly established until the vaccination lesion has healed, the immunity evidently spreading gradually through the skin from the site of inoculation. Vaccinated animals are susceptible to artificial reinfection subsequent to the primary inoculation, if such procedure is carried out before the original lesion has healed. But the lesion produced by the second inoculation is mild in character and usually of short duration. Obviously, the length of time elapsing between the two inoculations will determine the character of the lesion resulting from the second, the longer the duration of such period, the milder the type of lesion resulting from the second inoculation.

In an experiment designed to test the foregoing statements, we vaccinated 19 susceptible lambs in the right flank. On the seventh day, all of these animals showed well-developed lesions at the site of inoculation and were vaccinated in the left flank. In all of these lambs, we observed that the original vaccination lesion evolved naturally but that the lesions were healed on the 17th day after treatment, while the second inoculation developed only to the pustular stage, the vaccination site being clean nine days subsequent to treatment. The control animals, four in number, developed typical, severe lesions which healed 26 days after inoculation. The results of this experiment not only demonstrated that the partial immunity resulting from the first inoculation markedly reduced the severity and duration of the lesions from

the later inoculation but indicated that the second infection reduced the duration of the disease resulting from the first inoculation. This result indicates that the vaccination of infected animals in the field may be of practical benefit.

There is a considerable difference of opinion among investigators as to the duration of the immunity following vaccination. Carré[22] reports that animals recovering from a natural attack remain immune for two and one-half years, while vaccinated animals are susceptible after one year. Glover[9] states that animals remain solidly immune to artificial exposure for at least eight months subsequent to vaccination, while Lanfranchi[6] claims that the immunity is not always of long duration but may lapse after five to eight months.

In a controlled experiment, with seven animals which had been vaccinated approximately 13 months previous to reinoculation with potent virus, we observed that five (547, 548, 552, 549 and 551) were still immune to the disease. (See table III.) None of these animals showed the formation of either papules or vesicles following inoculation. Two others (550 and 553) developed very mild (+) lesions of the disease. The three controls contracted typical cases of the disease.

Inoculation of virulent scab emulsion into the scarified skin of the lips and flanks of young lambs, dropped by ewes which had been vaccinated against contagious ecthyma at the end of the second month of pregnancy, within 72 hours after birth, resulted in the development of a severe form of the disease in all tested animals and in the controls. The lambs were not vaccinated until they had suckled their mothers at least once. The results of this experiment indicate that the lamb inherits no immunity from an immune mother nor are there any protective substances contained in her milk.

Several investigators, notably Aynaud,[2] Theiler,[7] Glover,[9] and Schmidt and Hardy,[14] have done a good deal of experimental work with the immunization of susceptible lambs. Hatziolos,[16] in an outbreak of contagious ecthyma occurring in Greece, reported the successful vaccination of 5,226 susceptible sheep, stating that these animals remained healthy despite a prolonged retention on infected pastures. Schmidt and Hardy[14] reported the vaccination in the summer of 1931 of some 200 lambs and 77 kids in a field experiment where 300 lambs and 70 kids were left as controls. Approximately 80 per cent of the control kids later developed the natural disease, while the vaccinated kids remained free from the infection. The control lambs did not develop the disease.

TABLE III—*Contagious ecthyma immunity test. Exposure of vaccinated lambs to sheep virus approximately one year after vaccination, August 2 to August 11, 1933.*

LAMB	VACCINATED	AGE (MOS.)*	TREATMENT AUGUST 2, 1933	OBSERVATIONS		RESULT
				AUGUST 9, 1933	AUGUST 11, 1933	
547	6–23–32	16	Inoculated on scarified skin of lips with sheep contagious ecthyma virus in 1:100 dilution	Dry scab, flaky, thin. No inflammation	Normal	Negative. Immune
548	6–23–32	16		Dry scab, flaky, thin. No inflammation	Normal	Negative. Immune
552	6–23–32	16		Dry scab, flaky, thin. No inflammation	Normal	Negative. Immune
549	7–25–32	16		Dry scab, flaky, thin. No inflammation	Normal	Negative. Immune
550	7–25–32	16		Dry scab, some inflammation. Few dried pustules C. E. +	Dry scab; slight inflammation. C. E. +	Very mild "take." C. E. +
551	7–25–32	16		Dry scab, flaky, thin. No inflammation	Normal	Negative. Immune
553	8–10–32	16		Pustules dried up; slight inflammation. C. E. +	Dry scab; slight inflammation. C. E. +	Very mild "take." C. E. +
554 (Control)	.....	4		Many pustules. Swelling; severe inflammation	Pustules; moist scab; lips swollen, inflamed. C. E. ++++	C. E. ++++
555 (Control)	.....	4		Many pustules. Swelling; severe inflammation	Pustules; moist scab; lips swollen, inflamed. C. E. ++++	C. E. ++++
556 (Control)	.....	4		Many pustules. Swelling; severe inflammation	Pustules; moist scab; lips swollen, inflamed. C. E. ++++	C. E. ++++

*Age at time of exposure to sheep virus.

The next spring (1932), the present writers undertook field experiments on a much larger scale. Five ranches, where the disease appeared annually and usually in a rather severe form, were selected for this work. These ranches are located from 14 to more than 100 miles from each other and are typical of the sheep-ranching country of this part of Texas.

The motivating idea behind these experiments was to ascertain whether or not vaccination offered a practical, dependable means of protecting the lamb from sore mouth infection and consequent screw-worm damage during the hot summer months. In other words, we wanted to know if early vaccination of the susceptible lamb would carry it safely through the sore mouth season and deliver a good healthy lamb at weaning time in early October. If the lamb could be protected until weaning time, such protection would avert a tremendous loss for the sheep owners of western Texas. At that time we were not interested primarily in whether or not such vaccination immunized the lamb for longer than about six months.

In connection with the use of the vaccine in the field, it should be borne in mind that the virus contained in the vaccination scab, which drops on the range, keeps the infection alive on the premises. This fact obviously limits the use of the vaccine to animals on ranges where the disease is already prevalent.

The plan of the work carried out was the same in all cases. Approximately one-half of the total number of lambs on each ranch were vaccinated, the others being left as controls. The vaccinated lambs were run in pastures where they did not have contact through the wire fences with the control animals, such an arrangement precluding any possibility of the control lambs contracting the infection from vaccinated animals.

On each ranch the lambs were vaccinated as they were "marked." (In this region, "marking" is the term used to designate castration, docking and ear-marking.) These operations usually are carried out when the lambs are from three to eight weeks of age, but many younger lambs which may be included in the round-up also are marked. Consequently, the lambs in these experiments included animals from a few days of age up to animals approximately eight weeks old.

The technic of vaccination was the same on all ranches, the vaccine used being from a sheep strain of virus. The skin was scarified with a small "scratcher" made by soldering three sharp phonograph needles through a small cylinder of galvanized iron

at an angle of approximately 45 degrees. The needles were about 3/16 inch apart and parallel. Consequently the scarification consisted of three separate scratches, 3/16 inch apart and approximately one inch in length.

Immediately after the lamb was "marked," the skin of the flank was lightly scarified and a drop of vaccine was rubbed thoroughly into the scarified area with a small, stiff brush. The lamb then was dropped and the operation repeated on the next animal. Upon completion of the vaccination of all the lambs in each group, they were turned into the permanent pasture with their mothers.

Approximately two weeks after the vaccination had been completed on each ranch, several vaccinated lambs in each pasture were rounded up and inspected for "takes." In every case it was found that the animals had well-developed scabs at the point of inoculation. After this preliminary inspection, monthly visits were made to each ranch until the sore mouth season was ended in the region where these ranches are located. The vaccinated lambs were subject to ordinary range conditions at all times throughout the duration of these experiments.

The results of this work are given in table IV. It will be noted that a total of 7,884 lambs were vaccinated on these five ranches and that 10,173 lambs were left untreated as controls. The vaccinations were carried out during late March and early April on four of the ranches and during the last week in May on the fifth. Screw-worm flies were active on all of these ranches at the time of vaccination, particularly the one where the animals were treated on May 26.

The final results obtained in these experiments show that only 30 (.38 per cent of the 7,884 vaccinated animals contracted the disease subsequent to vaccination, while 6,667 (65.3 per cent) of the controls later developed the infection. In the final column of table IV it will be noted that the 30 vaccinated animals which contracted the disease showed only a very mild (designated by +) type while all of the controls developed a severe (++++) form.

As a consequence of the excellent results obtained from the foregoing experiments during the sore mouth season of 1932, it was decided, in a meeting of Experiment Station and Live Stock Sanitary Commission officials, to enlarge the scope of the experiment for the present year by making the vaccine available to the ranchmen at a nominal cost. Pursuant to this decision, meetings

TABLE IV—*Summary table of field vaccination experiments with contagious ecthyma, March 14 to August 29, 1932.*

RANCH	LAMBS*		TREATMENT	RESULT
	NUMBER	AGE		
A	2,511	2 days to 2 months	Inoculated on scarified skin of belly with 1 cc sheep contagious ecthyma vaccine 120 B, March 14-18, 1932	Sore mouth + 7
	5,204 (Controls)	2 days to 2 months	None	Sore mouth ++++ 2,852
B	782	3 days to 2 months	Inoculated on scarified skin of belly with 1 cc of sheep contagious ecthyma vaccine 157, April 1-6, 1932	Sore mouth + 15
	847 (Controls)	3 days to 2 months	None	Sore mouth ++++ 847
C	2,021	4 days to 2 months	Inoculated on scarified skin of belly with 1 cc of sheep contagious ecthyma vaccine 157, April 1-3, 1932	Sore mouth + 5
	2,012 (Controls)	4 days to 2 months	None	Sore mouth ++++ 1,684
D	2,338	2 days to 3 months	Inoculated on scarified skin of belly with 1 cc of sheep contagious ecthyma vaccine 157, April 4-8, 1932	Sore mouth + 1
	1,855 (Controls)	2 days to 3 months	None	Sore mouth ++++ 1,157
E	232	2 months	Inoculated on scarified skin of belly with 1 cc of sheep contagious ecthyma vaccine 157, May 26, 1932	Sore mouth + 2
	255 (Controls)	2 months	None	Sore mouth ++++ 127

*Total vaccinated lambs, 7,884, of which 30 developed sore mouth (+).
Total unvaccinated lambs, 10,173, of which 6,667 developed sore mouth (+ + + +).

were held throughout the sheep and goat region in which the results of the experiment were described and it was announced that the vaccine would be prepared for any ranchmen who were interested in vaccinating their sheep. In response to this information, orders for vaccine began pouring in until, by the end of February, we had demands for more than a million doses of vaccine.

The vaccination started about the first of December and continued, with more or less regularity, until the end of April. All told, 1,339,200 doses of vaccine were sent out, with the great bulk of the lambs being vaccinated during the month of March and the first two weeks of April. There is no question that approximately 1,500,000 lambs and kids were vaccinated, inasmuch as many more animals were vaccinated from each bottle than the dosage on the label indicated.

While all the ranchmen who vaccinated their animals have not been interviewed, information received from many of them demonstrate that the results are highly successful. The annual sore mouth season is practically ended at the present date and we have received no reports of vaccinated animals contracting the disease. In all probability some vaccinated animals have developed sore mouth, but the number is so small as to provoke no comment on the part of the sheep-owner.

In our field experiments last year and in the reports from ranchmen this year, it was noted that a small number (5 to 10 per cent) of the vaccinated lambs showed a very mild type of lip lesion about ten days subsequent to vaccination. Inasmuch as the natural disease is confined to the skin and many experiments have demonstrated that the induced lesions remain confined to the area where the inoculation is made, it follows that these mild lip lesions result from the animal transfering the infection from the vaccinated flank to the lips through biting or licking. In observations on literally thousands of flank-vaccinated lambs, we have observed only a very few severe cases of lip lesions developing subsequent to vaccination.

SUMMARY

The filtrability of the virus of contagious ecthyma of sheep and goats is demonstrated, but it is noted that the results of filtration are not always uniform. This finding confirms the results obtained by other investigators of the same disease.

Experiments with several animal species show that only sheep and goats seem to be susceptible to the disease.

The disease is prevalent throughout the sheep and goat-producing regions of western Texas and is of great economic importance.

Animals passing through an attack of the disease and those which have recovered from experimental inoculation possess a high degree of immunity, but it is not known definitely how long this immunity lasts.

During 1932, controlled field experiments demonstrated that a practical means of preventing the disease had been evolved through vaccination of the young lamb prior to the seasonal occurrence of the disease. Of 7,884 lambs vaccinated, 30 (.38 per cent) developed mild lip lesions subsequently, while 6,667 (65.53 per cent) of 10,173 control lambs contracted the disease in a severe form.

Results to date of the vaccination of more than 1,339,200 lambs, during the present year, indicate that vaccination is practical, economical and dependable.

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THE VETERINARY SITUATION IN ENGLAND

Some idea of how much the veterinary situation in England has improved lately may be gained from the following paragraph quoted from a letter recently received from Professor Sir Frederick Hobday, Principal and Dean, Royal Veterinary College, London, England:

> I take this opportunity to enclose the prospectus of the course of study at this College, together w'th a pamphlet issued by the Incorporated Associations of Head Masters and Head Mistresses of Public Secondary Schools and the Ministry of Labour, setting forth the prospects of the profession for the British graduate (male and female, respectively). It may interest you to know that we have 39 women graduates in the profession and 74 women students at this College. In England, there is practically no veterinary surgeon out of employment—male or female—and there is such a demand for the Army and Public State Service appointments that it is almost impossible for veterinary surgeons who have large practices to obtain assistants.

It would appear that veterinary medicine has made quite an appeal to the fair sex in England. The enrollment of 74 women students in one veterinary college causes all sorts of conjectures in the mind of one who recalls that during the past year the enrollment of one young lady in one of our American veterinary colleges was the cause for considerable newspaper comment.

Because of the scarcity of veterinary surgeons throughout many of the country districts of England, the Merionethshire Agricultural Association Committee has asked its agricultural organizer to interview the headmasters of the county secondary schools with a view to asking more pupils to proceed to a course of veterinary education, says the *Veterinary Record*. The Committee will consider applications for financial assistance toward the expenses of such a course.

Any Confederate veteran may fish in any county in South Carolina without obtaining a license, says the *American Field*.

12th International Veterinary Congress
New York—August 13-18, 1934

SOME SYMPTOMS AND LESIONS PRODUCED BY STOCK-POISONING PLANTS*

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A listing of the stock-poisoning plants of New England and a description of the symptoms and lesions produced by them would, without doubt, be of particular interest to this association. Unfortunately our information regarding most of the eastern plants is not sufficiently well supported by carefully planned and executed experimental studies.

For many years, the Department of Agriculture has been operating in the West a station for the study of poisonous-plant problems. In this work which, until 1930, was under the supervision of the late Dr. C. D. Marsh, the primary object has been to determine what plants are poisonous for live stock and under what conditions poisoning occurs. As the work has progressed, it has been found necessary to go into greater detail and to determine what changes take place in the toxicity of the various portions of the different plants during their growth, as well as to find out just how susceptible the different species or kinds of domestic animals are to being poisoned by them. As new poisonous-plant problems have been taken up for study, the Station has been moved from time to time and is now located in central Utah.

The experimental work done by the Department, and, we might add, that done by the state experiment stations, has been very largely in states west of the Mississippi River and with western plants. In other words, it has been done where the great bulk of the poisonous-plant problems arise.

In attempting to solve some of these problems, many species of plants have been fed experimentally to animals, more particularly to sheep, cattle and horses. Although the work is far from complete, the cases of poisoning thus produced have furnished considerable information regarding the symptoms and lesions caused by the plants worked with. What I say is based largely on the experimental work at our own stations. An attempt will be made, however, as far as possible, to apply the results to New England plants and local conditions.

Obviously, it will be impossible to discuss or even mention all

*Presented at the fourth annual meeting of the New England Veterinary Medical Association, Boston, Mass., November 6-7, 1933.

the plant species that have been worked with. In order to cover as much ground as possible, it will be desirable to group them in some way. Perhaps a grouping on the basis of the most characteristic symptoms shown by poisoned animals will serve our purpose.

Peculiarities of Poisonous Plants

First of all, I should like to call attention to certain differences and peculiarities of poisonous plants that have to be taken into consideration in their study. Many, perhaps the great majority of poisonous plants, cause poisoning when they are eaten in a sufficient quantity in a single feeding, or within a relatively short time—as during a single day. Such plants as the mountain laurel, sheep laurel, water hemlock, and choke cherry come in this class. On the other hand, many plants have cumulative effects and cause poisoning after they have been eaten in relatively small, subtoxic quantities at frequent intervals over a considerable period of time. The "locos" are an outstanding example of this type of poisoning. Other examples are oak leaves, horse tail, some of the ferns, and the poisonous ragworts. Some of these we will touch on later. Suffice it to say that there is overlapping of these two groups, some plants acting in both ways.

Another peculiarity which must be considered with plant poisons—one which frequently makes a diagnosis difficult—is a so-called latent period that occurs with some plants. Cases of this kind occur in both of the groups we have just mentioned. As an illustration I might cite two plants. We have in the Southwest, along the lower Rio Grande, in Texas, and in Mexico, a plant known to the natives as *coyotilla*. The most poisonous part of this plant is the fruit, or more particularly the seed, which is in a berry-like fruit about the size of a large pea. This plant is responsible for the poisoning of many sheep and goats. It also poisons pigs and sometimes calves. Even children, who, out of curiosity, have eaten the fruit, have been severely affected. After eating a relatively small quantity of these berries, symptoms may not appear for days, or even weeks. In one carefully controlled experiment, a sheep given a single feeding of a toxic dose appeared to be perfectly normal for 18 days and then became ill, showing typical symptoms of the poisoning produced by this plant. Chickens poisoned by it have gone as long as 42 days after being fed and then have become almost completely paralyzed in the legs. After symptoms once appear, they develop with rapidity and reach their most pronounced stage within a relatively short time.

Cumulative Effects of Ragwort

Among those plants which have cumulative effects, we might cite the latent period which often accompanies ragwort, or *Senecio*, poisoning. Animals fed on this weed daily for from two to three months or longer, and when apparently unaffected and turned into pastures, have gone from one to four weeks before symptoms of poisoning were noted. These then develop rapidly.

In contrast to these cases in which symptoms are delayed, we have plants which produce effects almost immediately after they have been taken into the stomach, even of ruminants. It has frequently been reported that cattle have become ill within a few minutes after being turned into fields of sorghum and Johnson grass. These cases, of course, are due to hydrocyanic acid produced by the plant. The same thing is true of the poisoning by cherry leaves, which, I understand, occurs frequently here in New England. When a sheep or cow is given a toxic dose of hydrocyanic acid by mouth, as a drench, it will show effects within from one to two minutes and may be dead within considerably less than half an hour.

Now let us consider the poisonous plants a little more in detail and, in so doing, group them on the basis of what has seemed to be the most prominent effect, or the effect which would be particularly noticed by the stockmen. Any such a system is, of course, open to objections. The symptoms produced by many plants are of such a nature that they might almost equally well fall into any one of two or more groups.

The most outstanding effect produced by many plants is a more or less profound depression. The sick animal, if on its feet, stands with the feet drawn close together, its head held very low, the ears drooped, and the animal showing a very pronounced disinclination to move. If lying down, its head will be held around to its side or rested on the ground. Such an animal, even if there is no indication of coma, will often refuse to make any attempt to get to its feet when disturbed and may pay no attention to surrounding objects. As a rule, the temperature, pulse and respiration are not seriously affected during the early part of the illness. Later, especially in fatal poisoning, the pulse will usually become fast and weak and the respiration labored. In those cases which end in death, the depression and weakness become more and more pronounced and the animal dies quietly. In such a case, one is very liable—I was going to say, almost certain—to find some very pronounced and more or less localized lesions. I can best point out what I mean by a few examples.

Greasewood Poisoning

We have in the semi-arid regions of Utah, New Mexico and Nevada, especially where there is a moderate amount of alkali in the soil, and the water table is not very far beneath the surface, a plant commonly known as greasewood, and to the botanist as *Sarcobatus vermiculatus*. This plant, which poisons many sheep, contains oxalates and potassium. An animal may eat considerable quantities of the leaves of the plant day after day without any injury. However, if it consumes more than a certain quantity within a minimum length of time, it will be fatally poisoned. In from two to seven hours after consuming the plant, the animal will appear depressed and it may be somewhat salivated.

An autopsy of such an animal will reveal little. There may be a slight congestion of the mucosa of the abomasum and the ileum, and sometimes the lungs are congested. On microscopic examination of the various tissues, little of significance will be noted except in the kidneys, and this is just what one finds in cases of poisoning by oxalates. Here we find a severe and acute parenchymatous nephritis. The epithelial cells, especially of the convoluted tubules, are swollen, greatly degenerated and the tubules themselves packed with calcium oxalate crystals.

This plant does not occur here in New England. However, rhubarb leaves have been suspected of poisoning pigs. As the leaves contain oxalates, one might reasonably expect the effects to be similar to those caused by the greasewood. Certain other plants here also contain oxalates, but these are very rarely, if ever, eaten by animals in sufficiently large quantities to poison them.

Cocklebur Poisoning

Another plant which produces severe depression and very often death is the ordinary cocklebur (*Xanthium* species). In this plant, the poisonous substance is in the seed. When the seeds germinate, their two portions, or cotyledons, swell and elongate and become the first two leaves. These contain the same poison as was present in the seed. They frequently grow so as almost to cover the ground in restricted areas. If eaten in sufficient quantities by animals, as they frequently are by pigs and sometimes by cattle, poisoning occurs. Symptoms develop in from about eight to 40 hours after the plant has been eaten. The animals are greatly depressed, the pulse frequently rapid and weak and the respiration is labored. With pigs, there may be

some vomiting. The animals usually become weak and die quietly.

The prominent macroscopic lesions noticed in animals that have died and were autopsied have been a severely congested or inflamed mucous membrane in the stomach (that is, in the fourth stomach of cattle and sheep), a less pronounced congestion of the mucosa of the intestine, and a congestion of the liver. Other tissues are usually anemic. With pigs and sheep we have found the livers more or less spotted or nutmegy, while in cattle, they have had a more or less bluish cast. The walls of the gall-bladder and the main bile-ducts have been thickened by an edematous infiltration.

The livers present a very interesting microscopic picture. Every one of the hepatic lobules is a mass of blood. The central lobular veins are obliterated by the hemorrhage. The normal arrangement of the hepatic cells into cords radiating from the central lobular vein has entirely disappeared. The hepatic cells of the central portion of each lobule are necrobiotic if not necrotic. They are separated and such cells as are left are scattered throughout the mass of blood. The only recognizable hepatic cells left are those near the periphery of the lobules. The general appearance is of an animal that has bled to death, the blood having lodged largely in the liver.

The kidneys in these cases are injured less than the liver. One finds some swelling and granular disintegration of the epithelial cells of the tubules and some of the cells show necrobiotic changes.

Milkweed Poisonous for Sheep

Another plant that produces this pronounced depression is a milkweed of California, known as the woolly pod milkweed, or *Asclepias eriocarpa*. This is responsible for sheep losses. Besides the depression, the plant causes a rise in body temperature and a fast, weak pulse. The principal gross lesions are congestion of the lungs, kidneys and portions of the alimentary tract. Microscopically, one finds accumulations of serum and masses of leucocytes beneath the epithelial layer of the various stomachs, and more or less necrotic changes in the epithelium itself. In fatal cases, where the animals linger along five or six days, one is liable to find areas in the first, second, and third stomachs where the mucous membrane is represented by masses of granular débris, the result of cell disintegration. The kidneys of such cases are more or less degenerated, the changes being those of acute parenchymatous degeneration.

The ground seeds of corn cockle are particularly injurious to chickens and produce symptoms of depression similar to those described. Here the principal lesion is in the crop and sometimes the proventriculus. It usually consists of a severe congestion and an edematous swelling of the wall of the crop and the surrounding tissues. This is accompanied by pronounced hemorrhage into the swollen tissue and a laking of the red blood corpuscles. The whole thickened mass often becomes necrotic and it has been known to protrude through the ruptured skin of the neck and eventually to slough off.

Nausea Produced by Certain Plants

Many plants cause symptoms of nausea in cattle, sheep and goats. The nausea is apparent from the frequent regurgitation, an excessive flow of saliva, and more or less profuse vomiting. In mild cases this may be about all one will observe. In more severely poisoned animals, other symptoms of course will develop.

In New England a number of members of the so-called *Heath* family of plants occur. These fall in this group. The ones which may poison animals in this section are the mountain-laurel, sheep laurel or lamb-kill, and possibly the bog-rosemary, rhodora and the great laurel. These all contain a similar poisonous substance called andromedotoxin, and consequently produce the same effects. Sheep, goats and cattle are poisoned by them. As a rule, symptoms develop in about six or seven hours after the plants have been eaten, although they may be apparent in as little as three hours; or they may not be apparent for as much as 20 hours or more. Depending on the severity of the poisoning, the illness may last from one to two hours to as much as four or five days.

Usually the first thing to be noticed in poisoned sheep or goats is a tendency to swallow very frequently, or one may notice, especially in sheep, frequent movements of the neck and throat, indicating repeated regurgitations and re-swallowing of the rumen content without any normal chewing of the cud. About the same time the animals will show evidence of an increasing flow of saliva, and may be somewhat dull or depressed. These effects grow rapidly more pronounced and the animal will usually vomit. Sometimes this vomiting is very severe and large quantities of material will be gotten rid of. After a time the animal will grow weak, will step about uneasily, eventually lie down, and may appear to be in a semi-comatose state. If sufficiently affected it may be unable to get to its feet again for four or five days. In some cases the respiration becomes labored. In mild poisoning the pulse and body temperature are as a rule unchanged.

Symptoms of poisoning in cattle are very similar to those shown by sheep, although the salivation is not so pronounced.

Now and then poisoned sheep will develop a pneumonic condition. Whether this is a mechanical pneumonia, due to the drawing of regurgitated material down the esophagus, or is a direct effect of the toxic substance, is not entirely clear. Such cases develop a temperature that is somewhat higher than normal.

In those cases which end fatally, the principal lesions are in the kidneys. Here there is severe congestion, edema, and some hemorrhage, as well as a pronounced and acute parenchymatous nephritis affecting all the tubules and leading to necrobiotic changes. There is also some cloudy swelling of the hepatic cells of the liver.

In animals which develop a pneumonic condition, the lungs are congested and there is a severe pulmonary edema accompanied with an invasion of the tissues by polymorphonuclear leucocytes. The injury centers about the bronchi and has the appearance of a mechanical pneumonia.

Sneezeweed Produces Nausea

I should like to mention another plant which causes severe nausea. I refer to the so-called sneezeweed *(Helenium hoopesii)*, of the Rocky Mountain regions. This is related to the sneezeweed of the East, a plant which I am told gets into western Massachusetts. It causes very severe losses of sheep in portions of Utah. We have seen many sheep in a band sick at one time, all with their heads held high in the air and attempting to hold the regurgitated material, which was running in quantities out of the corner of their mouths. Sneezeweed is poisonous also to cattle in some areas and produces the same effect on them.

This plant may poison by single doses or by repeated small doses, the latter being the usual way under range conditions. It ordinarily takes about three weeks after sheep or cattle start grazing on this plant before symptoms appear.

Some sheep never show the vomiting effect but become weak. Such animals can stand but a short time. If found lying down and made to get up, they at first appear fairly normal. Within a very few minutes they become uneasy, keep shifting their weight, and commence to tremble and shake. This trembling increases in violence and the animal very soon either lies down or its legs crumple under and it falls. At this time the respiration and pulse rate are very rapid. Rest very soon restores the animal to an apparently normal condition.

If deprived of sneezeweed the trembling type of cases rapidly recovers. The vomiting type probably rarely recovers. Although few animals that vomit die when first ill, they do die one after another, later on, during the winter or when subjected to hardships. Most such cases simply appear as unthrifty animals, and many develop a cough. They lose flesh and when any particularly unfavorable condition develops, they may die. Sheep that have been poisoned may vomit frequently for months after they have eaten the weed, especially when they fill up on green feed.

Poisoned animals that vomit profusely often develop extensive adhesions and areas of consolidation throughout the lungs. This, I take to be due to mechanical irritation. In addition to the lung lesions, usually one finds on autopsy some congestion of the kidneys and of the mucous membrane of certain portions of the alimentary tract. Under the microscope the most constant change is in the liver tissue. The hepatic cells are either much swollen and the intralobular capillaries compressed, or else the cells are compressed and the capillaries distended. The cells are cloudy, frequently edematous, and often fatty. In some cases there is a small cell infiltration. Evidences of blood stasis are apparent. In the central veins there are more or less granular débris and numerous leucocytes, and usually loosened and free hepatic cells are found in the blood-stream.

Cases of acute poisoning can be produced by this plant by feeding to a sheep a sufficient quantity of it within a short time. The symptoms resulting are of the depression type, and the principal lesions center about the part of the alimentary tract where the ingested plant lies. As a rule, it will be found lying in the rumen not far from the opening of the esophagus. At this point the walls of the first and second stomachs will be congested, greatly thickened by coagulated serum, and there will be a mass of coagulated serum on the serous surface, more particularly in the rumino-reticular groove. In addition, the walls of the stomachs in this region will contain great numbers of leucocytes, some of which will be necrotic. Obviously, the toxic substance is very severely irritating and kills the tissues with which it comes in contact in a sufficiently concentrated form. It then penetrates the tissue itself, killing the cells as it goes through the wall.

Colorado and Texas Rubberweed

Because of the serious losses of sheep that are caused by two closely related plants—the Colorado rubberweed and the bitter rubberweed of western Texas—I wish to call attention to them.

Their action on animals is similar to that of the sneezeweed, although the nausea is much less pronounced.

We have come to look upon the characteristic effects of certain plants as a more or less violent trembling of any muscles which are subjected to use. Two of these should be mentioned because of the seriousness of the results that they sometimes produce. These are the white snake-root of the Ohio Valley and of the Carolinas, and the rayless goldenrod of New Mexico, Texas and Arizona. The illness caused in animals by white snakeroot is commonly called "trembles" or "slows," while that which is due to the rayless goldenrod in the Southwest is usually spoken of as "alkali disease." When human beings are affected, the term "milksickness" is also used. White snakeroot grows in New England but as far as we know never causes losses in this region.

After eating these plants for some time the animals show signs of weakness, and will tremble violently on slight exertion, the trembling being most noticeable in those muscles that are being used. Thus, sometimes while chewing their food, the muscles of the jaw will show marked trembling. Such animals are weak and will frequently assume a kneeling position. The weakness grows more and more pronounced until the animal is unable to get to its feet at all and it dies quietly. Illness may last for some time. Usually such animals are constipated, the feces being hard and dry. During the early stages of the illness, the temperature, respiration and pulse remain normal. In severe illness the respiration is labored, the inspiration is slow, a pause follows the inspiration, and the expiration is forced. There is usually a distinct odor of acetone present about the animal.

One of the most peculiar things about these plants is the fact that the poisonous substance is excreted with the milk, being largely associated with the fats, and the disease may be passed on to the calf or person using these substances. Human cases of the so-called milksickness are characterized by extreme depression, obstinate constipation, subnormal temperature and a distinct odor of acetone. Recovery is exceedingly slow and any exertion during convalescence is very liable to cause a fatal relapse. Cases of milksickness practically always occur in individuals or groups of persons using the milk, cream, or butter from one or two cows that have for some reason been feeding on one of the plants just mentioned. Consequently they occur almost entirely in rural communities.

More or less severe fatty changes occur in the livers of animals dying from trembles and of human beings affected with milksickness. This, I think, is the most characteristic lesion accom-

panying these diseases. There are evidences of irritation around the bile-ducts and the gall bladder and the bile is usually very dark and viscid. Other organs show minor injury but these appear to be secondary to those in the liver.

Constipation and Diarrhea

A number of plants, such as certain species of the larkspurs, or *Delphinium*, cause severe constipation; others, such as some of the milkweeds, have just the opposite effect. A group of plants which are of interest because of their effects are the oaks. Oak leaves—at least, those of certain species—are responsible for rather serious losses of cattle. Probably cattle in New England never eat them in sufficient quantities to be seriously affected. The so-called shinnery oak, a species that grows in the sandy regions of New Mexico and western Texas, is a good example of the oaks that do cause trouble. Because of general conditions there are periods when the cattle have little besides oak to eat. After feeding almost exclusively on this plant for about two or three weeks, they become severely constipated, the coat is rough, the nose becomes dry and soon forms a scabby encrustation that cracks and peels off, and the animals will be found standing around the watering places in a very dejected manner. Usually the head is held with the nose extended forward. After a little, instead of obstinate constipation there is an equally severe diarrhea. Mastication ceases and very shortly the animal is passing almost pure mucus. This often is present in large white coagulated masses.

Unless found before the diarrhea sets in, death is almost certain to result. The animal simply grows continually weaker and passes out of the picture without a struggle.

The gross lesions found on autopsy are partly those which accompany starvation. Pronounced accumulations of serum are found beneath the skin, ascites is often pronounced, the mesentery may be very greatly thickened and of a jelly-like consistency, and a similar edematous condition may be found in the other serous surfaces of the body cavities. When thickened, the mesentery is usually studded with small hemorrhages due evidently to ruptured blood-vessels. Usually the mucosa of the fourth stomach and the small intestines is inflamed and the kidneys are congested.

Microscopically, the kidneys show the most significant lesions. They are edematous and the glomeruli are shrunken or compressed. The tubules are distended and many casts are often found in them. The epithelium of the tubules is sometimes flat-

tened and always more or less degenerated. Some of the distended tubules resemble cysts.

One other type of poisoning which is of interest here is that caused by the various sorghums, and, more particularly, by cherry leaves. These cases are due to the plants containing substances which under proper conditions are capable of producing hydrocyanic acid. The symptoms and lesions produced are the same as those caused by the prussic or hydrocyanic acid itself.

Many plants apparently act directly on the central nervous system. The lesions which most of these produce are not very definitely known. Even with plants which have been given considerable study, such as the locos, the lupines and the *coyotillo*, little is known about the relationship between symptoms and lesions.

Plants Which Produce Spasms

Two of our native plants produce violent spasms. These are the water hemlock (species of *Cicuta*) and certain species of whorled milkweeds. A whorled milkweed, *Asclepias galioides*, will be mentioned because it produces very severe spasms and is related to a form which, although it is very rare, occurs here in New England.

The effects of the whorled milkweed are characteristic. The first evidence of illness is a weakness or staggering which appears in from three to 20 hours after the plant has been eaten. Very soon after symptoms first appear, the animal will fall and may throw itself about very vigorously. Very often the head is raised into the air and slammed back onto the ground with great force. Very soon the head will be thrown back in a position of opisthonotonos. It will then almost at once be violently drawn down to the breast into a position resembling emprosthonotonos. The feet, however, are drawn up to the body. Almost at once the muscles relax for a moment and then the legs are moved rapidly in running-like motions. Soon these cease, the animal relaxes for a moment and the whole process starts over again. These convulsions continue at frequent intervals but after some time they grow less and less violent until the animal dies. In one case, 59 such spasms occurred in a 30-minute period. Most cases end fatally. Naturally, the pulse is very rapid and the respiration labored. During the period of spasms the body temperature is high. Rectal temperatures of 110° F. have been recorded in sheep.

An autopsy on these cases reveals very little. A microscopic study of the tissues brings out little that would appear to account for the symptoms. Some organs, as the myocardium of the ventricles, are congested. Naturally one would look for the principal effects to be in the central nervous system. There is some capillary

congestion and some edema in various portions of the brain and spinal cord, with a little hemorrhage by diapedesis about some of the smaller blood-vessels. There is also a considerable distention of the perivascular and pericellular lymph-spaces.

The Purkinje cells of the cerebellum show various stages of fatigue effects, apparently depending on the violence and duration of the spasms. In one case we found the bulk of these cells had reached a stage of complete exhaustion. This, however, I take to be a result and not the cause of the spasms.

Cirrhosis of the Liver

A group of plants which causes liver cirrhosis has in recent years attracted considerable interest not only in America but in various other parts of the world. These plants are commonly known as ragwort, or groundsels, and to the botanist as species of *Senecio*. Several species of this group of plants—and probably some belonging to other genera—produce the same, or similar effects. Recently Dr. F. W. Schofield, in Ontario, has associated liver cirrhosis in horses with the feeding of alsike clover.

From a historical standpoint the investigations of this subject are very interesting. You all know something of the pictou cattle disease of Nova Scotia. For many years the Canadian government condemned and slaughtered herds of cattle in which this disease appeared, on the grounds that it probably was contagious. It was not until 1905 that it was shown by Dr. W. H. Pethick to be due to feeding the cattle for a prolonged period on hay containing *Senecio jacobaea*.

Senecio poisoning occurs in both cattle and horses and can be produced in sheep. It can be produced by feeding a large quantity of the plant in a single feeding, or by feeding small quantities daily over prolonged periods. The length of time required for symptoms to develop depends on the quantities fed daily.

After a prolonged course of feeding, the test animal may appear entirely normal and be turned out to pasture, and then some two to four weeks later be found to be sick.

In both horses and cattle, two types of cases occur. In both, the first thing noticed is a lack of appetite and a depressed attitude. There may be a peculiar, sweetish and penetrating odor about the animal, and there may be some yellowish discoloration about the eyes and other visible mucous membranes. The membranes usually grow more and more discolored. The urine is frequently very dark in color.

With cattle, the nose becomes caked over, and a dirty yellowish discharge often comes from the nostrils. Serous accumulations

sometimes develop under the skin of the head, around the ears and on other parts of the body. If there are old brands or other areas of scar tissue on the skin, the yellowish serum will sometimes actually ooze through the tissue. These areas are evidently unpleasant and the animals sometimes rub or press them against any convenient object. In one type of cases, these symptoms grow more pronounced and the animals become weaker and die quietly. In the other type of cases, the animals become uneasy and walk about a great deal, and will often viciously charge any person or moving object that comes near them.

In our experiences, most affected horses have a depraved appetite, refuse all hay and grain, but chew boards and dirt. After a preliminary period of depression they usually become uneasy and from then until they go down, they walk almost continuously in an aimless manner. At first they avoid objects but later they will walk into trees, fences, or any object in their way. If it is a fence, they will push ahead in a frenzied, persistent manner and often work themselves into a perspiration. They are then hard to control or handle. In such a stage, they will walk through a wire fence or any other object that they can force their way through. All affected horses are said to die sooner or later.

In such animals—both cattle and horses—the livers are seriously affected, the condition depending on the length of the illness. They are firm and tough, often have a greenish, or sometimes a mottled, appearance, and are usually severely congested. In cattle, the gallbladders are often very greatly enlarged and surrounded with coagulated serum, and the bile is usually very mucilaginous. Often in cattle, too, the mesentery—especially that portion in the folds of the colon—is very greatly thickened, due to the presence of quantities of coagulated serum; and sometimes the mucosa of the abomasum is in a similar condition and of a jelly-like consistency. Microscopically, the liver of an animal poisoned by *Senecio* may be very hemorrhagic. The whole cellular arrangement is upset, the hepatic cells are in all conceivable stages of degeneration and repair, and new-formed bile-ducts and new growth of connective tissue in the more prolonged cases are very abundant. One finds all conceivable combinations of destruction of hepatic cells and excessive growth of connective tissue. In extreme cases, there may be no normal hepatic cells left and each lobule may be surrounded by a mass of scar tissue. Even small groups of hepatic cells may be cut off by this new growth.

These few cases will, I think, be sufficient to show that the symptoms and lesions produced in animals by poisonous plants are extremely varied. The accumulation of information is a slow

process. While some progress has been made, what has been learned is only a beginning. Our knowledge of the effects of the great bulk of stock-poisoning plants is fragmentary and very incomplete. The data on hand are of interest but not yet sufficient to warrant more than very general opinions as to the relationship between the lesions and the symptoms exhibited by the poisoned animals.

Iowa Joins the List

The Iowa Veterinary Medical Association has taken its place with the other state associations that have affiliated with the A. V. M. A., being the 41st state to take this action. On the basis of its membership in the A. V. M. A., Iowa will have three votes in the House of Representatives at its first meeting on Wednesday afternoon, August 15, at the Waldorf-Astoria, in New York City.

Gradually the list of states that have not affiiliated with the national organization is being reduced until now only six are left that either have taken no action toward this end, or else have not apprised the A. V. M. A. headquarters of such action if it has been taken. The six states that now comprise the unaffiliated list are: Arkansas, Mississippi, New Hampshire, New Mexico, West Virginia and Wyoming. All of the other states are expected to be represented at the meeting in New York, with the exception of Idaho, which has no state association.

Becker Features Saint Bernards

Bob Becker, of the editorial staff of the *Chicago Tribune*, has been giving a series of broadcasts on dogs from station WGN and allied stations this summer. Becker's radio programs on dog subjects have caused widespread comment for their sympathetic and understanding handling of dog stories and tips on dog problems. A recent feature was an interview with Hans Blaser, a native Swiss, who unfolded the intensely interesting story of the Saint Bernard breed. His chat with Becker was followed by a vivid dramatization of the death of Barry, greatest of the Saint Bernards, who saved so many lives in the lonely Alpine passes.

12th International Veterinary Congress
New York—August 13-18, 1934

THE EFFECT OF DIET ON THE COURSE OF EXPFRIMENTAL COCCIDIOSIS INFECTION IN THF CHICKEN*

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The experiments reported here were undertaken to test the value of the protein and vitamin-A content of a diet in the control of experimental infections of chickens with *Eimeria tenella*. The work of Allen[1] had led her to conclude that a high-protein and high-vitamin (A and B) diet produced a chronic course of *Eimeria tenella* infection characterized by (1) lowered oöcyst production during the first five days, (2) low mortality rate, (3) less hemorrhage, and (4) failure to regain good physical condition within 18 days after oöcysts first appeared. On the other hand, a low-protein, low-vitamin diet appeared to be conducive to high production of oöcysts early in the infection and high mortality, but rapid recovery of the surviving birds. Any modification of the course of coccidial infection through variation of diet seemed of such importance from both the practical and scientific points of view, that further work on the influence of protein and vitamin content of diets on coccidiosis infections was considered desirable.

In the present experiments the rations were planned to eliminate as many variable factors as possible. Five rations† were made which differed from one another only in those constituents providing protein and vitamin A, to form the following diets:

I. High-protein, high-vitamin-A content,
II. High-protein, low-vitamin-A content,
III. Medium-protein, high-vitamin-A content,
IV. Low-protein, high-vitamin-A content,
V. Low-protein, low-vitamin-A content.

Throughout the text, these diets are referred to by the corresponding Roman numerals, and groups of experiment birds are identified by the Roman numeral corresponding to the diet they were fed.

The formulae for the rations are given in table I and the chemical analysis of each ration appears in table II.

*This work was supported partially by a Fellowship established by the Chas. M. Cox Company, of Boston, and the National Oil Products Company, of Harrison, N. J., for the year 1932-1933. Received for publication, November 21, 1933.

†These rations were planned by Mr. W. S. Baker, of the Eastern Grain Company, and were made up under his supervision.

TABLE I—*Composition of experimental diets.*

CONSTITUENT	DIET I	DIET II	DIET III	DIET IV	DIET V
Yellow corn meal	44	0	44	44	0
White corn meal	0	44	0	0	44
Wheat bran	10	10	10	10	10
Standard wheat middlings	5	5	5	5	5
Red dog flour	0	0	12	17	17
Ground rolled oats	5	5	5	5	5
Alfalfa leaf meal	2	2	2	2	2
Calcium carbonate	2	2	2	2	2
Edible bone meal	0	0	0	3	3
Fortified cod-liver oil	⅛	⅛	⅛	⅛	⅛
Soy bean meal	5	5	5	5	5
Dried skim milk	7	7	7	7	7
Meat scrap (60% protein)	10	10	4	0	0
Fish meal (60% protein)	10	10	4	0	0
Totals	100⅛	100⅛	100⅛	100⅛	100⅛

High protein calculated = 24.2 per cent protein.
Medium protein calculated = 18.9 per cent protein.
Low protein calculated = 14.5 per cent protein.

The differences in vitamin-A content of the rations have been achieved by the use of yellow corn meal in the high-vitamin diets, and of white corn meal in the low-vitamin diets. The protein content has been varied by the amounts of meat scrap and fish meal employed. Steamed bone meal has been added to the rations from which meat scrap and fish meal were removed to hold the total ash, calcium and phosphorus content nearly constant. Milk was left at the same level in all the rations since it has been suggested that varying quantities of milk have an effect on the course of coccidiosis infections. The development of a number

TABLE II—*Chemical analysis of experimental diets.*

CONSTITUENT	DIET I	DIET II	DIET III	DIET IV	DIET V
Moisture	8.60	8.50	8.70	8.80	8.80
Crude protein (N x 6.25)	25.60	25.50	20.70	17.00	16.60
Crude fat	5.20	4.50	5.10	4.90	4.40
Ash	8.20	8.20	5.70	6.50	6.00
Crude fiber	3.80	3.50	4.00	4.00	3.50
Nitrogen-free extract	48.60	49.80	55.80	58.80	60.70
Total calcium calculated as Ca	2.33	2.37	2.03	1.53	1.78
Total phosphorus calculated as P	1.21	1.19	1.00	0.85	0.94

of cases of "slipped tendon" among chicks on the high-protein, low-vitamin ration, and to a less extent among those on the high-protein, high-vitamin diet, which interfered with the normal growth of the birds involved, made it necessary to remove many of them from the experiments. The diets involved have a high total phosphorus content which is thought (Payne, Hughes and Leinhardt[2]) to be an etiological factor in the development of the "slipped tendon" condition.

TABLE III—*Lynnfield control groups—average weight (gm) per bird.*

WEEK	GROUP I		GROUP II		GROUP III		GROUP IV		GROUP V	
2	105		105		99		88		91	
3	176		181		173		136		145	
4	247		272		261		204		207	
5	357		363		363		269		266	
6	493		476		505		380		360	
7	686		680		689		524		493	
8	797		794		822		658		573	
9	814		910		987		729		689	
	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀
10	1176	1040	1114	1060	1264	1103	1052	924	816	887
11	1287	1154	1276	1188	1403	1225	1216	1077	958	1009
12	1460	1228	1318	1287	1588	1361	1375	1142	1083	1165

To secure the normal growth curves of uninfected chickens on the five experimental rations, a pen of 50 chickens was raised on each of the diets. These birds were kept in batteries at the Wirthmore Experimental Farm for a period of twelve weeks. The chickens were weighed at weekly intervals from the second through the twelfth week. The average weights in grams for the five groups are presented in table III. Weekly examinations were made of fecal material collected at random from each cage. During the eleventh week, a very light infection with *E. acervulina* was found among the birds on diet II, and during the twelfth week a similar light infection among the birds on diet V. At the last three weighings, the birds were divided according to sex.

A preliminary experiment (series I) was performed in which different amounts of a suspension of *E. tenella* oöcysts were given to birds on diets I, III and V. It was desired by this method to determine the number of oöcysts necessary to produce a severe infection without too high a mortality. The doses ranged from 0.5 cc to 2.0 cc of the same suspension of oöcysts.

The material was introduced into the esophagus by means of a sterile medicine-dropper. The birds were fed the experimental diets for three weeks before infection. They were kept on autoclaved sawdust litter in cages which had been sterilized. The food was autoclaved for 20 minutes at 15 pounds pressure, and drinking water was taken from the hot-water tap. At the time of infection, the birds were placed on wire trays which were changed every second day, as were also the feed and water dishes. This procedure, although not eliminating reinfection completely, prevented any gross reinfection, since oöcysts were not found in the droppings after the 22nd day. It has been demonstrated in carefully controlled experiments (Tyzzer, Theiler and Jones[3]) that oöcysts from a single infection may be produced over a period of 19 days. The mortality resulting from these infections is shown in table IV.

TABLE IV—*Mortality in series I after infection with graded doses of E. tenella oöcyst suspension.*

Diet	Dosage								Dead on Each Diet	
	0.5 cc		1.0 cc		1.5 cc		2.0 cc			
	Inf.	Died	Inf.	Died	Inf.	Died	Inf.	Died	Total	Per Cent
I	4	1	4	1	.....	.....	2	0	2	20
III	4	1	4	1	.....	.....	2	1	3	30
V	2	0	2	0	2	1	2	1	2	25
Totals	10	2	10	2	2	1	6	2	7	

Other criteria of severity of infection, such as time of appearance, duration and amount of hemorrhage; time of resumption of cecal function; duration of oöcyst production; and average number of oöcysts per gram of droppings were taken into consideration. (See table V.)

From these results it is evident that even the smallest dose produced some fatal infections, while the largest dose, which contained approximately four times as many oöcysts, yielded no corresponding increase in mortality. In the present instance the introduction into the crop at one time of equal numbers of oöcysts failed to produce infections of uniform severity and the feeding of varied doses yielded no correlation between numbers of oöcysts introduced and severity of infection. Since only those sporozoites reaching the cecums are able to establish themselves in an *E. tenella* infection, the severity of infection depends on the

number of sporozoites reaching the cecums and not on the number of oöcysts fed.

It has been pointed out previously (Tyzzer *et al.*[2]) that there is great wastage of *E. tenella* sporozoites, since they appear in large numbers in the feces after feeding heavy doses of the oöcysts. From the results of feeding graded doses of oöcysts, it would appear that unequal quantities of material containing sporozoites reach the cecums in a given period of time in different birds, with consequent greater loss of sporozoites in the feces of some birds than in others. Difficulties in estimating dosage had been encountered in early experiments on the effect of age on *E. necatrix* infections (Tyzzer *et al.*[3]). The procedure adopted in later tests with *E. necatrix* was used in the present experiments for subsequent *E. tenella* infections. This procedure consisted of mixing the infective material evenly with the mash which was given in the morning after the birds had fasted overnight. The birds were allowed to eat the mash for a period of two hours, when the feed and water dishes were removed and replaced by sterilized dishes with uninfected mash and fresh water. This permitted continuous introduction of oöcysts into the crop over a period of time and tended to equalize the chances of sporozoites reaching the cecums.

The results of the preliminary experiment (series I) may be summarized as follows:

Dosage: Approximately equal numbers of birds died after being fed 0.5 cc, 1.0 cc and 2.0 cc of the same suspension of oöcysts, when the material was introduced into the esophagus by pipette.

Deaths from coccidiosis: No correlation between numbers of deaths and diet was evident. It is realized that the numbers used in this experiment were very small.

Hemorrhage: Blood appeared in the droppings on the same day in all three groups. It was greatest in amount in group I and persisted over the longest period.

Weight: The condition of certain of the birds in group V was extremely poor. The average gain in weight of the group was far behind that of the control group, and also much less than that of the inoculated groups I and III (see table IV). It was thought the difference might be due in part to the autoclaving of the diet, since the vitamin content of their diet (V), already low, might have been further unbalanced by the heating process. It was decided therefore to eliminate this variable in future experiments, and to feed the mash without autoclaving.

A comparison of the condition of the birds in the three groups showed six of eight surviving birds in group I to be plump and in good condition, while two were judged to be in only fair condition. Of seven surviving birds in group III, four were in good condition, and three fair; of six survivors in group V, one was in good condition, four thin, and one very thin.

The birds were killed 28 days after inoculation. No oöcysts were found in the cecums at autopsy in any of the three groups.

It will be seen from the description of this preliminary experiment that the birds in group V were in very poor condition, although they had suffered no greater mortality than the two other groups. It is doubtful whether they ever would have regained a growth rate normal for the V diet.

The results of this experiment (series I) are tabulated in table V.

TABLE V—*Series I. Course of E. tenella infection in chickens on three experimental diets.*

	Diet I		Diet III		Diet V	
	Inoculated	Control	Inoculated	Control	Inoculated	Control
Chicks	10	50	10	50	8	50
Aver. weight (gm), 2nd week	100	105	91.5	99	69.5	91
Aver. weight (gm), 7th week	636	686	473	688	272	493
Average gain (gm) in weight	536	581	381.5	589	202.5	402
First appearance of blood in droppings	5th day		5th day		5th day	
Last appearance of blood in droppings	12th d'y		8th day		8th day	
Inoculated chicks dead from coccidiosis	2		3		2	
Mortality (%)	20		30		25	
Average number of oöcysts per gram of droppings for 7th, 8th, 9th and 10th days after infection	972,860		668,800		818,180	
Average amount of fecal material (gm) per day	21		28		11	

A second experiment (series II) was carried out to determine the course of coccidiosis in birds first infected with *E. tenella* after seven weeks on the experimental diets. This is an age at which natural infections may be acquired on the range. Ten chicks (five males and five females) from group I, and ten, also equally divided as to sex, from group V were brought to the

laboratory from the control pens at Lynnfield after seven weeks on the diets. They were placed in sterilized cages on wire mesh and were continued on their respective rations (non-autoclaved). They were fed a suspension of *E. tenella* oöcysts, mixed with the mash as described above.

On the fifth day, five birds on diet I (high-protein) and four on diet V (low-protein) showed hemorrhage. On the following day, all the birds in both groups were passing bloody droppings, but the amount of blood was noticeably more copious in the group I pens. On the seventh day, three birds on diet I and three on diet V died, and on the eleventh day, a fourth bird on diet I succumbed. All of the surviving birds in both groups were extremely sick. Hemorrhage continued in all the pens until the tenth day after infection. A normal cecal dropping (from a bird on diet V) was noticed for the first time on the 17th day, indicating the first resumption of normal cecal function.

As there appeared to be very little difference in the reactions of the two groups of birds to the initial infection, it was decided to give the surviving birds a second somewhat lighter infection to discover if a more chronic course of the disease could be produced in one group than in the other. The second feeding of oöcysts was given 13 days after the first. The wire bottoms were removed from the cages and the chicks were allowed to run on a litter of autoclaved alfalfa leaf meal. This provided opportunity for self-reinfection from the floor of the cage. Observations were made on individual birds at intervals. No hemorrhage was observed in any of the birds, and there were no deaths from this second experimental infection. Oöcysts were discharged over a period of 18 days in group I on the high-protein diet, and over a somewhat longer period in group V on the low-protein diet. An adventitious infection with *E. acervulina* complicated the observations to a certain extent at this time, and the birds of both groups were again placed on wire platforms to eliminate this infection.

Two weeks later, observations on all birds had become negative, and a third heavy feeding of *E. tenella* oöcysts was given. On the sixth day following this last feeding, all birds in group I showed some hemorrhage which was severe in two. Only one bird in group V showed hemorrhage, and it was not severe. Oöcysts were produced by all birds of both groups. The birds were killed nine, ten and eleven days after this feeding, the condition of the cecums noted, and the number of oöcysts estimated. The results are given in table VI. If the presence of only degenerating oöcysts in scrapings of the cecal wall is taken as the

criterion of immunity in a bird, 50 per cent of the birds in group I (high protein) and 42 per cent of the birds in group V (low protein) developed immunity to a third infection.

TABLE VI—*Condition of cecums at autopsy of chickens in series II.*

GROUP	CONDITION OF CECAL WALLS	OÖCYSTS IN CONTENTS	OÖCYSTS IN SCRAPINGS OF CECAL WALLS
I	Normal....1 Walls thickened, contents normal...2 Cores present....2 Hemorrhage....1	Negative....1 Moderate to heavy infection....5	Rare, degenerate oöcysts....3 Numerous oöcysts....3
V	Normal....4 Walls thickened, contents normal...1 Cores present....1 Hemorrhage....1	Negative....3 Moderate to heavy infection....4	Degenerate oöcysts...3 Moderate numbers of oöcysts....3 Heavy infection....1

The weights of these birds, which were 15 weeks of age when killed, averaged, in group I, 1,893 grams for males and 1,423 grams for females; and in group V, 1,397 grams for males and 1,348 grams for females. A comparison with the weights of control uninfected birds at Lynnfield at the eleventh week of the experiment, when recovery from the second *E. tenella* infection was practically complete among the experiment birds, is made in table VII.

TABLE VII—*Comparison of weights (gm) of controls and E. tenella-infected birds at 11 weeks of age.*

	GROUP I HIGH-PROTEIN DIET		GROUP V LOW-PROTEIN DIET	
	MALES	FEMALES	MALES	FEMALES
Lynnfield controls (average per bird)	1287	1153	958	1009
E. tenella-infected (average per bird)	1375	1093	903	826
Difference in weight in infected birds as compared with control birds	+88	−60	−55	−183

From these figures, it appears that the males of group I suffered no loss of weight, but rather an increase in spite of their *E. tenella* infection, while the other birds all showed a loss which

was most marked in the females of group V. The results of this experiment (series II) are summarized in table VIII.

TABLE VIII—*Course of E. tenella infection in chickens on high-protein and low-protein diets.*

	GROUP I HIGH-PROTEIN DIET	GROUP V LOW-PROTEIN DIET
Chickens inoculated	10	10
Appearance of hemorrhage (after first infection)	5th day	5th day
Disappearance of hemorrhage (first infection)	10th day	10th day
Nature of hemorrhage (first infection)	Severe, copious	Moderate
Mortality (first infection) (%)	40	30
Immunity to third infection (%)*	50	42

*Based on presence of degenerating oöcysts in scrapings of cecal wall.

Since the mortality had been equally severe in groups on the high- and the low-protein diets among the chickens in series I and following the first infection in the older birds of series II, which had been longer on the diets, it seemed improbable that any significant difference in the mortality resulting from an initial *E. tenella* infection would be produced by the various rations. The next experiment, therefore, was planned to determine whether greater chronicity would be developed on certain of the rations rather than on others, and, if prolonged infections existed in all the groups, whether an equal degree of immunity to a subsequent massive infection would be developed.

Sixty day-old chicks were divided into six groups of ten each, one group being placed on each of the five experimental diets for four weeks prior to infection. The sixth group was placed on the group III ration (average-protein, high-vitamin) to serve as a laboratory control to check the effect of laboratory conditions on the weight curve, against conditions at Lynnfield where the other controls had been kept.

The chicks were kept in cages which had been sterilized, and were given autoclaved alfalfa for litter. Their food was not autoclaved. Fecal examinations were made from time to time, but no evidence of coccidial infection was found prior to the experimental inoculation.

A light initial infection of *E. tenella* was given after four weeks on the experimental diets by mixing a suspension of oöcysts with the mash. No hemorrhage resulted from this in-

oculation, nor were there any deaths. Oöcysts appeared in the droppings of all the birds after seven days. At this time, the birds contracted the *E. acervulina* infection which had appeared in the birds of the older groups then under observation in the same room. The birds were put on wire-mesh platforms, and continued observations were made on both cecal and small-intestinal droppings from individual birds. Infections with both species continued over a period of approximately three weeks.

At this time, a heavy second infective feeding of *E. tenella* oöcysts was given the five groups of birds. Bloody droppings appeared in all pens on the fifth day, and the hemorrhage was of equal severity in all pens. All the birds appeared droopy and sick. On the sixth day, there was one death in group I, none in group II, five in group III, three in group IV, and two in group V. After the eighth day, there was no further hemorrhage in group I, and none was observed in the other groups after the tenth day.

Following the method used by Allen[1] in her third series, oöcyst counts were made on the five groups for the first four days of oöcyst production. The counts are in direct contrast with those of Allen for the first five days, in that groups I and III, on the high-protein, high-vitamin diet, and average-protein, high-vitamin diet produced approximately twice as many oöcysts per gram of fecal material as did the birds on the low-protein diets, or the high-protein, low-vitamin diet. Our counts agree with those of Allen in showing a peak of oöcyst production on the second day. In estimating counts of total oöcysts produced, it should also be considered that the average number of grams of fecal material per bird for the first four days is approximately 100 per cent greater for birds in group I, and approximately 70 per cent greater for birds in group III than for birds in group V.

The mortality for the two high-protein pens (groups I and II) was 7.7 per cent, while that of the low-protein pens (groups IV and V) was 26.3 per cent. These figures are comparable with Allen's results, which showed a mortality of 5 per cent on the high-protein, high-vitamin diet and 23 per cent on the low-protein, low-vitamin diet. It should be noted, however, that the mortality rate in the present experiment was highest among birds in group III, on the normal-protein, high-vitamin diet, the mortality in this group being 45.4 per cent. If the protein content of the diet were the effective factor in controlling mortality, this group should be intermediate between the high-protein (I and II) and the low-protein (IV and V) groups.

The high-vitamin content of diets I and III might have been considered the factor favorable to greater oöcyst production, were it not for the low oöcyst count of group IV on the low-protein, high-vitamin diet.

The average weights per bird in each of the six groups during the first eight weeks of the experiment are tabulated by sex in table IX.

TABLE IX—*Average weights (gm) of chickens of series III which survived eight weeks.*

Week	Group I High-Protein High-Vitamin-A		Group II High-Protein Low-Vitamin-A		Group III Medium-Protein High-Vitamin-A				Group IV Low-Protein High-Vitamin-A		Group V Low-Protein Low-Vitamin-A	
							Controls					
	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀
1	74	70	75	78	68	68	73	69	60	65	65	64
2	124	111	125	130	107	107	118	106	82	95	105	94
3	210	181	233	230	180	168	200	184	123	145	160	143
4	316	266	345	335	263	249	288	239	176	214	210	202
5	411	362	460	425	337	331	393	330	251	280	280	266
6	454	421	503	470	382	371	500	460	269	321	330	311
7	629	575	625	578	531	507	712	580	343	398	385	398
8	779	665	753	688	650	617	845	705	469	447	455	464

A third infective feeding of *E. tenella* oöcysts was given to the surviving birds in the five groups 19 days after the second feeding, to test the immunity developed by the previous exposures. The birds were killed on the seventh day after their third infective feeding, and the cecums were examined for thickening of the walls, hemorrhage, presence of cores, and oöcysts either in the contents or in scrapings of the cecal walls. Birds showing no evidence of disease were judged immune. The percentage of immune chickens found in each of the five groups is shown in table X. The two high-protein groups showed less immunity than the two low-protein groups, and group III showed the highest immunity of all.

DISCUSSION

In considering the three series of experiments as a whole, the only consistent result which may be assigned to the effect of any diet is the failure of the infected, low-protein groups to gain in weight proportionately with their uninfected controls. In the first series, it was felt that autoclaving the diet had been deleterious to these groups in particular. In the second series, in

TABLE X—*Course of E. tenella infection in chickens on five experimental diets.*

	Diet I	Diet II	Diet III		Diet IV	Diet V
			Infected	Control		
Chicks inoculated	10	10	10	10	10	10
Average weight (gm), 1st week	72	73	68	71	63	65
Average weight (gm), 8th week	722	712	632	747	457	463
Average gain (gm)	650	639	564	676	394	398
Average gain of 50 controls at 8th week	796	794		822	658	573
Deaths from other causes	2	5	0	0	1	0
Appearance of hemorrhage (second infection)	5th day	5th day	5th day		5th day	5th day
Disappearance of hemorrhage (second infection)	8th day	10th day	10th day		10th day	10th day
Mortality (second infection) (%)	12.5	0	50		33.3	20
Immunity to third infection (%)	20	50	75		50	62.5
Average number of oöcysts per gram (first four days) of 2nd infection	1,532,750	832,000	1,659,050		699,550	833,400
Total droppings (gm) for first four days	259	215	214		237	127
Total number of oöcysts produced in first four days	397,000,000	179,000,000	355,000,000		166,000,000	106,000,000

which the rations were not autoclaved, the difference between the high- and low-protein groups was not so marked as in either the first or third series, but the birds were older when they received their first infection. In the third series, the weights of all groups fell somewhat below those of the Lynnfield controls. A comparison of the average weight at eight weeks of uninfected Lynnfield controls on diet III (medium-protein) with that of uninfected laboratory controls showed the Lynnfield controls to be 75 grams heavier. If this difference is taken as a constant value for the variation between all Lynnfield and laboratory groups, the two infected, high-protein groups compared favorably in weight with the uninfected groups. On the other hand, infected groups III, IV and V weighed 16, 27 and 7.5 per cent less than would be expected on the basis of the uninfected medium- and low-protein groups. The outstanding feature of these experiments was the good physical condition maintained by infected birds on the high-protein diets, a finding in direct contrast with Allen's results.

Hemorrhage appeared to be more severe in birds on the high-protein diets in the first two series. In the third series, however, this difference was not observed. Neither the time of first appearance of hemorrhage nor its duration appeared to be influenced by diet.

The mortality in the first two series was approximately the same for the high- and low-protein groups. In the third series, the high-protein groups had fewer deaths than the low-protein, and the highest mortality was in group III, on the medium-protein diet. This group also had the highest immunity among the surviving birds. However, immunity among survivors and mortality are not well correlated among the other groups of the series.

Chronicity of infection was not influenced by diet in series I in which the course of single infections was followed until oöcysts were no longer present in the feces. The birds on the low-protein diet were negative on the 19th day after the infective feeding, and the birds on the medium- and high-protein diets were negative on the 22nd day.

Immunity to reinfection among birds on high- and low-protein diets varied in series II and III. In series II, 50 per cent of the high-protein and 42 per cent of the low-protein group were immune to a third infective feeding of *E. tenella*. In series III, there was a marked difference in immunity to a third infective feeding between the high-protein, high-vitamin group (I) with 20 per cent immune, and the low-protein, low-vitamin group (V) with 62.5 per cent immune. The difference is not so striking when the two high-protein and the two low-protein groups are con-

trasted with each other, since both the high-protein, low-vitamin group (II) and the low-protein, high-vitamin group (IV) were 50 per cent immune. When corresponding protein groups of series II and III are averaged, 31.5 per cent of the birds on high-protein diets were immune to a third infective feeding, as against 52.4 per cent of the birds on low-protein diets. Since a slowly developing immunity implies chronicity of infection, these results furnish more definite indication of greater chronicity on high-protein diets than observations based on the course of single infections.

SUMMARY

Chickens were maintained on diets differing in their protein and vitamin-A content, and the effects of infection with *Eimeria tenella* were noted.

No differences in the course of *E. tenella* infections were observed which could be attributed to the vitamin-A content of the diet.

Chickens on high-protein diets maintained their weight at a level nearer that of their uninfected controls than chickens on a low-protein diet. High-protein diets may thus be considered as furnishing a reserve which prevents undue loss of weight as a result of coccidial infection.

Chickens on high-protein diets developed immunity to reinfection more slowly than birds on low-protein diets.

Oöcyst production on the basis of number of oöcysts per gram of fecal material was greater in chickens on high-protein diets than among birds on low-protein diets, and was still greater on the basis of total counts.

The amount and duration of hemorrhage and the mortality were not consistently correlated with diet in these experiments.

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It is always good when a man has two irons in the fire.

Francis Beaumont.

12th International Veterinary Congress
New York—August 13-18, 1934

STUDIES IN INFECTIOUS ENTERITIS OF SWINE

VIII. Isospora Suis N. Sp. in Swine*

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During the course of routine examinations of feces from swine, we occasionally encountered protozoan forms showing characteristics of the sub-family Isosporinae. Examination of the literature reveals the presence of Isospora in a number of mammals, birds, and some of the cold-blooded vertebrates but their occurrence in swine has not been recorded.

An experiment pig (3851) weighing about 35 pounds was encountered, which showed a considerable number of oöcysts in the feces. A few oöcysts had already reached the early sporoblast stage, while in the remainder the protoplasm was in the "ball" form. The oöcyst elimination is expressed by the number of oöcysts found in the average of 10 low-power fields made from centrifuged sugar preparations: July 26, 1932—126; July 27—82; July 28—68; July 29—25; July 30—3. These results indicate that the pig was encountered at the time of or shortly after the peak of the oöcyst elimination, *i. e.*, during the latter half of the infection.

The oöcysts obtained from these eliminations were placed in 2.5 per cent potassium dichromate in liter serum bottles and thoroughly aerated daily by passing compressed air through the medium. Nearly all such forms sporulated *in vitro*. Descriptive and developmental studies were made from oöcysts present in the feces of experimentally infected swine. The feces were collected from the pen-floors twice daily. In the recently collected preparations the oöcysts sometimes showed a granular protoplasm arranged in an irregular spheroidal form. In most of the oöcysts, however, the zygote had already reached the "ball" form (fig. 1). The zygote measurered about 16.5μ in diameter. In nearly all instances the zygote showed a slight clock and counter-clock movement. This movement was so sufficiently pronounced and frequent that in making photomicrographs it was necessary to shorten the exposure time by increased illumination and removal of the color filter in order to arrest motion. A structure simu-

*Received for publication, February 5, 1934.
†The coöperation of Dr. A. E. Brandt, of the Statistical Laboratory, in the preparation of the statistical data, is acknowledged with thanks, as are the helpful suggestions by Dr. E. H. Becker, Department of Zoölogy.

lating a polar inclusion, measuring about 2.8μ in diameter, is often present between the zygote and the oöcyst wall. At this stage the oöcysts were quite resistant to the influence of heavy sugar solutions, very little distortion or change of form taking place after exposure for three to five hours. After sporulation occurred in 2.5 per cent potassium dichromate, this was not the case; the oöcyst wall collapsed and adhered closely to the sporocysts (fig. 7).

The unsporulated oöcysts are subspherical in shape, possessing a double wall which measures about 1.5μ in thickness. The oöcyst wall is light yellow with a faint tint of brown. Measurements taken from 50 oöcysts revealed the data shown in tables I and II.

TABLE I—*Measurements of 50 unsporulated oöcysts Isospora suis.*

LENGTH		20μ	21μ	22μ	23μ	24μ	TOTALS
Breadth	18μ	4	2		2	1	9
	19μ		1	7	3	2	13
	20μ			12	7	7	26
	21μ					2	2
Totals		4	3	19	12	12	50

TABLE II—*Summary of measurements of unsporulated oöcysts Isospora suis.*

	LENGTH	BREADTH
Range	20 to 24μ	18 to 21μ
Mean	22.50μ	19.42μ
Standard deviation	1.16	.83
Coefficient of variation	.05	.04
Average form index	1.158	

After suspension and aeration in 2.5 per cent potassium dichromate for about 24 hours, some individuals showed an irregular arrangement of the protoplasmic granules, and variations in the size of the granules. A light zone appeared in the center of the mass (fig. 2). These changes preceded the cleavage into two sporoblasts (fig. 3). In about 24 hours, a great many of the forms had undergone complete cleavage, at which stage the oöcyst showed a spherical body comparable to a polar inclusion and frequently another smaller, irregular shaped structure, suggesting a residual body, also was present. When the preparations were from 27 to 36 hours old, the sporoblasts passed into the early

sporocystic stage (fig. 4). Each sporocyst presented at this time a heavy membrane without interruptions. At the same time the contents of the sporocysts showed faint outlines of the developing sporozoites.

By the method used, it required about four days in most instances for oöcysts, passed in the feces, to reach the infective stage as determined by microscopic examination (fig. 5). In the completely developed stage, each of the two sporocysts possesses a heavy double wall having a thickness of about 0.66μ. Thirty sporocysts taken at random and measured revealed the data showed in tables III and IV.

TABLE III—*Measurements of 30 sporocysts of Isospora suis.*

LENGTH		16μ	16.8μ	17.0μ	17.2μ	17.6μ	TOTALS
Breadth	10μ	6					6
	11μ	11		2			13
	12μ	3	1	4	1	2	11
Totals		20	1	6	1	2	30

TABLE IV—*Summary of measurements of sporocysts of Isospora suis.*

	LENGTH	BREADTH
Range	16μ to 17.6μ	10μ to 12μ
Mean	16.37μ	11.17μ
Standard deviation	.56	.74
Coefficient of variation	.034	.066
Average form index	1.465	

Each sporocyst contained a spherical granular body. After the second day, this sporocyst body decreased in volume by the progressive loss of the component granules, leaving a spherical hyaline body measuring about 3.3μ in diameter. Figure 6 shows this hyaline residual structure after many of the surrounding granules had been lost and in figure 9 this hyaline structure is shown after complete disappearance of the granules. Each sporocyst contained four sporozoites. No measurements are recorded of the sporozoites owing to the great pressure required to excyst them, thus producing distortion.

Completely sporulated oöcysts were washed several times in saline to remove the potassium dichromate used in the sporulating medium. These were then placed in artificial gastric juice com-

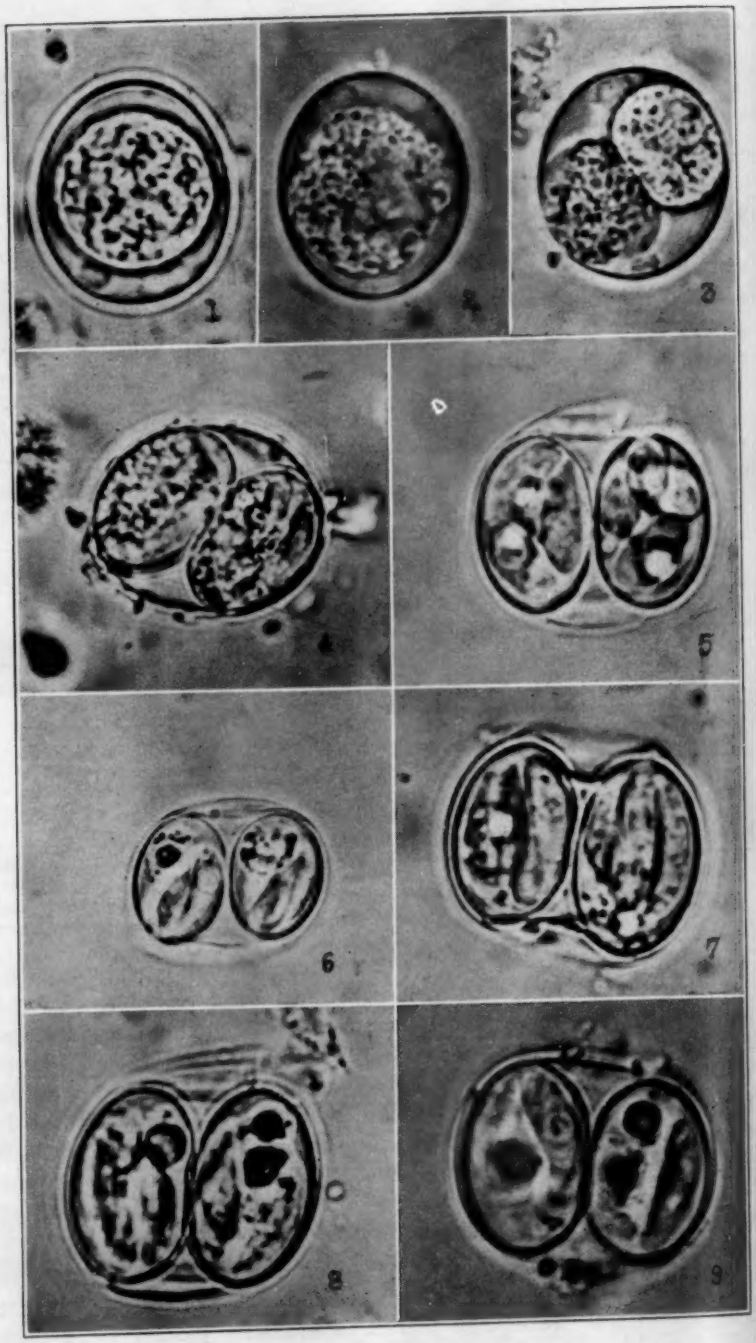

PLATE I

posed of 1 per cent hydrochloric acid and 2 per cent pepsin. These preparations were held at a temperature of 37° C. for five hours, being agitated several times during this period. The oöcysts then were centrifuged and the supernatant fluid poured off and fresh artificial gastric juice was added, the preparations being stirred and mixed, after which they were incubated again for 18 hours. At the end of this period, the suspensions were again centrifuged and the artificial gastric juice replaced by a mixture composed of 0.3 per cent sodium carbonate and 0.5 per cent pancreatin. After thorough mixing, the suspensions were incubated for 4 hours at 37° C. At the end of that period, the preparations were centrifuged again and a fresh mixture of sodium carbonate and pancreatin was added, followed by incubation for 17 hours at 37° C.

Then the suspensions were centrifuged and the oöcysts floated on the surface of sugar solution, harvested by means of a blunt glass rod and mounted on a glass slide under a square cover-glass. The cover-glasses were carefully and thoroughly sealed on all sides by means of "Deckglasszement." Then pressure was exerted on the cover-glasses by means of a flat rubber stopper in an attempt to rupture the cysts. The oöcyst wall was mutilated or broken in most cases but the sporocysts remained within the broken oöcyst wall, which adhered to the sporocysts (figs. 5 and 8). In only a few instances were sporozoites found outside of the cyst and these appeared distorted as a result of the severe pressure exerted. When sporulated oöcysts were treated with artificial digestive juices for shorter periods, the oöcyst walls were broken in fewer individuals. When untreated sporulated oöcysts were allowed to remain in a sugar solution like that used in the sugar flotation method, the oöcyst wall merely collapsed and adhered to the sporocysts (fig. 7).

KEY TO PLATE I

FIG. 1. Oöcyst with zygote in "ball" form (x 1400).
FIG. 2. Changes in zygote preparatory to cleavage (x 1400).
FIG. 3. Sporoblastic stage (x 1400).
FIG. 4. Development of sporocysts showing faint outlines of sporozoites. (x 1400).
FIG. 5. Infective stage; sporozoites fully formed (x 1500).
FIG. 6. Sporocystic residual bodies showing partial loss of granules and appearance of hyaline body or residue (x 1100).
FIG. 7. Segmented oöcyst held in sugar solution, showing collapse of oöcyst wall and its adherence to sporocysts (x 1500).
FIG. 8. Segmented oöcyst subjected to artificial digestive juices followed by pressure. Oöcyst wall ruptured but still surrounding the two sporocysts (x 1500).
FIG. 9. Showing complete disappearance of granules of sporocyst residual body, with hyaline structure remaining (x 1500).

EXPERIMENTAL INFECTIONS

Attempts to produce experimental infections were made about two months after the first series of cultures was obtained from a naturally infected pig. Four pigs about five months old were used. Possible previous infections with *Isospora suis* could not be ruled out. These subjects were held for 14 days prior to the administration of *Isospora suis*, during which time the feces were negative. At the time the sporulated preparation was fed, it was about two months old. Most forms appeared degenerated. The sporocyst contents were granular and contained vacuoles. Similar preparations of swine Eimeria *(E. scabra* and *E. debliecki)* showed only very slight degenerative changes after a similar lapse of time. The results of feeding this preparation of *Isospora suis* are shown in table V.

TABLE V—*Results of feeding sporulated Isospora suis oöcysts to four pigs (9-23-32).**

DATE	OÖCYSTS PER LOW-POWER FIELD			
	PIG 1	PIG 2	PIG 3	PIG 4
9–23–32	+	+	+	+
9–24–32	–	–	–	–
9–25–32	–	–	–	–
9–26–32	0	0	0	0
9–27–32	0	0	0	0
9–28–32	0	0	0	0
9–29–32	0	0	0†	0†
9–30–32	0	0	0†	0†
10– 1–32	0	0	0	3†
10– 2–32	1†	0	1†	1†
10– 3–32	0†	0	0	1
10– 4–32	–	–	–	–
10– 5–32	6.4†	0	1‡	1‡
10– 6–32	1†	1	1‡	0‡
10– 7–32	0	0	0	0
10– 8–32	0	0	0	0

*Six pigs held in the same barn were used as control animals. All remained negative.
†Slight diarrhea.
‡Constipation.

In order to obtain a more viable suspension and a greater number of this Isospora, oöcysts eliminated by pigs 1 and 4, shown in table V, were placed in potassium dichromate and aerated. When sporulated, a part of this preparation was fed to two non-infected pigs each weighing about 40 pounds. Both were free from Isospora infection for at least ten days previously, as determined by fecal examinations. In one pig a pronounced in-

fection occurred while in the other only a slight one was observed. The control pigs, not shown in the chart, remained negative. The results of the fecal examinations on these infected pigs are summarized in table VI.

Oöcysts of *Isospora suis* from pigs 5 and 6 were collected and sporulated. These were fed to five pigs for the purpose of studying the pathology in various stages of the infection. Unfortunately the only pigs available were subjects weighing about 80 pounds, in which previous infections could not be ruled out. Only low-grade infections resulted (table VII).

It should be noted that the first preparation obtained from a naturally infected pig was passed successively through three series of pigs shown by tables V, VI and VII. Another preparation of *Isospora suis* was subsequently obtained from a 35-pound pig brought in from the Institute pasture. This preparation was fed to three pigs from the same source, all of which were not infected at the time, as determined by daily fecal examinations

TABLE VI—*Results of feeding sporulated Isospora suis culture to two pigs.*

DATE	OOCYSTS PER LOW-POWER FIELD	
	PIG 5	PIG 6
10–11–32	0	0
10–12–32	0	0
10–12–32*	0	0
10–13–32	0	0
10–14–32	0	0
10–15–32	0	0
10–16–32	–	–
10–17–32	0	0
10–18–32 A.M.	0†	1†
P.M.	0†	5†
10–19–32 A.M.	–†	7†
P.M.	0†	11†
10–20–32 A.M.	2	27
P.M.	2	39
10–21–32 A.M.	2‡	90‡
P.M.	2‡	35‡
10–22–32	1‡	42‡
10–23–32	–	–
10–24–32	0	5
10–25–32	0	2
10–26–32	0	0
10–27–32	0	0
10–28–32	0	0
10–29–32	0	0

*Sporulated Isospora culture fed.
†Diarrhea.
‡Constipation.

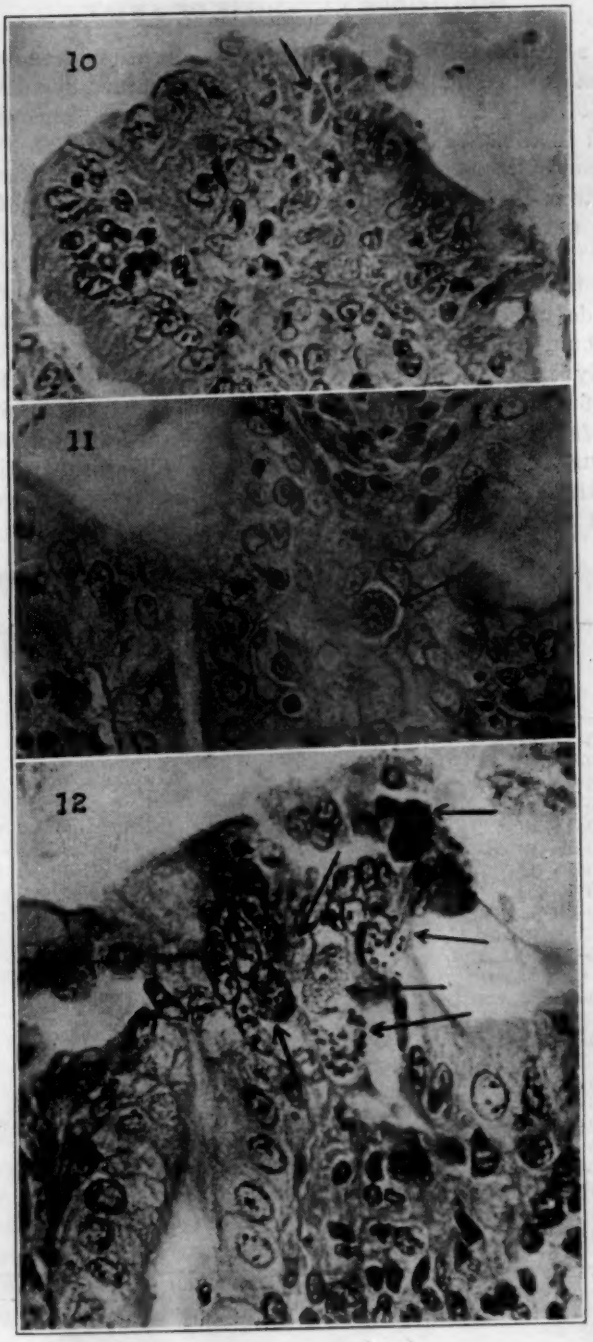

PLATE II

over a period of six days. All three pigs developed low-grade infections. Oöcysts were present in the feces six to eight days after feeding and were eliminated for seven days in one subject. Anorexia and diarrhea, followed by constipation, also were observed in these pigs. A large amount of mucus and epithelial cells was present in the feces during the infection. Control pigs kept in the same building remained negative.

Pathology

The feeding of sporulated *Isospora suis* to swine always resulted in infections, although slight in some pigs as indicated by the elimination of newly-formed unsporulated oöcysts. The low-grade character of most of the experimental infections might be attributed to the rapidity with which forms held *in vitro* degenerated and also to a partial immunity on the part of the swine used, it being impossible to eliminate possible previous

Table VII—*Results of feeding sporulated Isospora suis to five pigs.*

Date	Oöcysts per Low-Power Field				
	Pig 7	Pig 8	Pig 9	Pig 10	Pig 11
10-31-32	0	0	0	0	0
11- 1-32	0	0	0	0	0
11- 2-32	0	0	0	0	0
11- 3-32*	0	0	0	0	0
11- 4-32*	0	0	0	0	0
11- 5-32	0	0	0	0	0
11- 6-32	—	—	—	—	—
11- 7-32	0	0	0	0	0
11- 9-32	0†	0	0	0	0
11-10-32		0	0	0	0
11-11-32 A.M.		5	0	0	0
P.M.		10	0	3	2
11-12-32		8	3	5	8†
11-13-32		—	—	—	—
11-14-32		14†	3	3	
11-15-32			1	1	
11-16-32			—	—	
11-17-32			0	0	

Isospora suis fed per os.
†Killed to obtain tissues.

Key to Plate II

Fig. 10. Showing disturbance of surface epithelium and presence of merozoite (x 625).
Fig. 11. Macrogametocytes of *Isopora suis* in mucosa (x 525).
Fig. 12. Macrogametocytes of *Isopora suis* in mucosa with epithelial reactions (x 625).

infections. Aside from the diarrhea, anorexia and subsequent constipation, no other clinical manifestations were observed in controlled, experimentally infected swine.

Pig 7, fed *Isospora suis*, was killed six days later to study the parasite tissue relations and the pathology produced. Scrapings from the mucosa taken at intervals of six inches showed a few free merozoites in the lower third of the duodenum. Nearly all preparations from the jejunum contained motile merozoites. All sections of the ileum contained these forms.

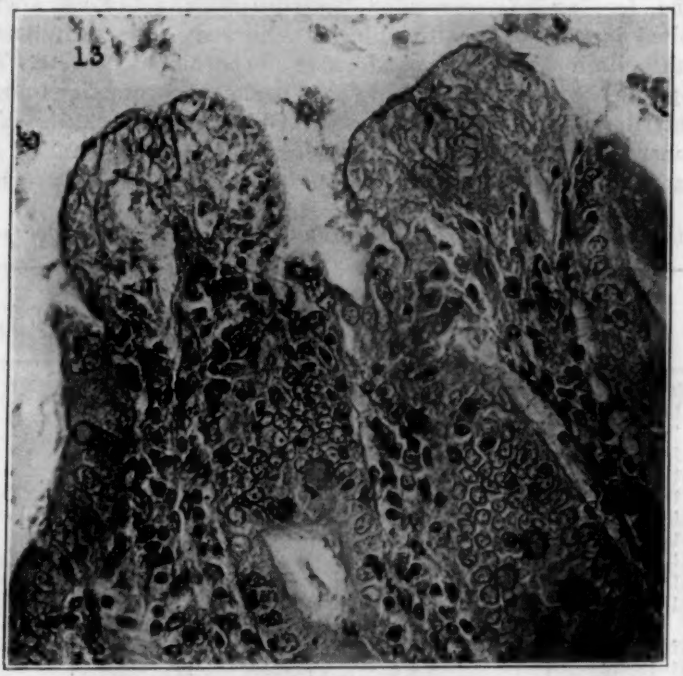

FIG. 13. Loss of surface epithelium and necrosis of substantia propria of the terminal portion of villi (x 550).

Histologic preparations taken at intervals of approximately six inches throughout the small intestine contained individual or groups of trophozoites, merozoites and schizonts in the cells. Figure 10 shows a merozoite in the midst of an area of retrogression and degeneration adjacent to the lumen. A subepithelial and interstitial inflammatory reaction appeared in connection with the attack on the epithelium in the beginning. In many instances the invaded epithelial cells migrated towards a subepithelial position, but often both the invaded epithelial cell and

the parasite appeared to be undergoing retrogressive changes and instead of migrating to a subepithelial position the parasitized cells desquamated. In tissues studied from a series of pigs rendered partially or totally resistant to *Eimeria scabra* it was found that when the merozoites invaded the epithelial cells both cell and parasite succumbed and disintegrated. During this activity many of the epithelial cells desquamated instead of migrating to a subepithelial position which occurred when susceptible pigs were fed infective oöcysts of *Eimeria scabra*. In addition, the *substantia propria* of the pigs infected with *Isospora suis* showed degenerative changes which advanced to the necrobiotic stage (fig. 13).

Pig 11 was killed nine days after the first feeding of *Isospora suis*. In some wet preparations made from surface scrapings and contents of the jejunum and ileum, a few merozoites and macrogametocytes were found, as well as a very early oöcyst stage. Scrapings from the colon and rectum were negative but the contents contained fully developed oöcysts. In the latter part of the duodenum and the first two feet of the jejunum, individual forms and groups of macrogametocytes were present together with mucosal destruction (figs. 11 and 12).

Pig 8 was killed eleven days after the first feeding of infective *Isospora suis*. Scrapings from all parts of the small intestine were negative, although a few newly-formed oöcysts were present in the rectal contents. When this fact is considered in the light of the oöcyst eliminations of the preceding days, it appears that the tissue invasion had stopped. The small intestine of this pig showed the same typical surface desquamation and rather characteristic necrosis of the *tunica propria* of the tips of the villi in which the nuclei were entirely or in part destroyed, leaving only a reticulated structure. A concomitant inflammatory reaction in the balance of the wall also was noted.

In view of the apparent partial immunity possessed by the pigs used and the behavior of the parasitized cells, it is not felt that the series should be used as a basis for constructing the intra-host stages of the Isospora in question, nor should it form the basis for a description of parasite-tissue relations in the various stages. The pathologic changes are confined to the small intestine, commencing with the first two or three feet of the duodenum. Grossly the changes are those of a non-specific catarrhal enteritis. The microscopic changes consist of the destruction of the epithelium of the tips of the villi, never involving the epithelium lining the crypts except near the intestinal lumen. The *substantia propria* of the tips of the villi is destroyed, leaving a reticular honeycomb

structure devoid of cells and nuclei in the final stage. Interstitial inflammatory reactions of varying degrees of intensity are present with a marked eosinophilic infiltration, the myelocytic type of cells predominating. These changes are not associated with gross hemorrhage.

The pathology suggests that *Isospora suis* infections are not fatal but that they produce intestinal alterations and destruction sufficiently pronounced to retard growth for a time and may be in part responsible for unthriftiness. This is supported by the clinical observations. In experimentally infected subjects, diarrhea commenced on about the sixth day after sporulated forms were fed. The diarrhea continued for three or four days, followed by constipation. The feces became very hard and dry and were passed in the form of hard pellets or masses usually covered with a grayish yellow mucus and fibrin exudate. Fecal examinations revealed a very large content of mucus. These manifestations are accompanied also by anorexia and unwillingness to move.

Attempted Inter-Host Infections

To determine whether or not the Isospora recovered from swine is host-specific for swine, viable sporulated Isospora were fed to four guinea pigs on two different occasions. No infections resulted in the guinea pigs, although swine fed from the same material became infected. The feces of the guinea pigs were negative for more than three weeks following the ingestion of such sporulated oöcysts. Two attempts to infect dogs by means of the swine Isospora were unsuccessful. *Isospora bigemina* was fed swine but no infections resulted, the feces remaining free from oöcysts for nearly a month following the ingestion of viable sporulated forms.

Discussion and Summary

1. The sporulation time of *Isospora suis* is relatively short. Unlike the Eimeria of swine, *Isospora suis* degenerated very rapidly and sporulated preparations could not be held long *in vitro*. Because of this fact it became necessary to use promptly available pigs which were older than desired and in which possible previous infections since birth could not be precluded.

2. Experimental *Isospora suis* infections produce anorexia and diarrhea followed by constipation.

3. Surface desquamation and necrosis of the terminal portion of the *substantia propria* of the villi of the small intestine, beginning about two feet from the pylorus and extending up to the last two or three feet of the ileum, are noted. A pronounced

interstitial inflammatory reaction with eosinophilic infiltration of the myelocytic type occurs.

4. *Isospora suis* and its various stages of sporogony are described.

5. The rôle of this protozoön and the Eimeria as pathogens in swine should not be overemphasized. If they are found during the occurrence of a hemorrhagic condition of the intestines, the coccidia should be identified or isolated and subjected to animal tests before attributing the hemorrhagic condition of the intestinal wall to the coccidia present. In such cases encountered by us, the coccidia present did not produce bloody stools when isolated and fed to pigs. Anorexia and diarrhea followed by constipation were observed in the experimentally infected pigs.

6. Attempts to infect guinea pigs and dogs with *Isospora suis* failed. The swine were not infected by feeding *Isospora bigemina* from the dog.

Congress Notes

Prof. Dr. W. Steck, who is on the program as a reporter on "Infectious Mastitis," has been chosen by the University of Berne, Switzerland, to be its official delegate.

Dr. F. C. Minett, of London, will do the honors for three institutions: The Royal Veterinary College of London, the University of London and the Research Institute in Animal Pathology of the Royal Veterinary College of London. He is also a reporter on "Infectious Mastitis."

Prof. Dr. G. K. Constantinescu and Prof. Dr. G. Nichita have been designated official delegates of the Veterinary Faculty of Bucharest, Roumania. Dr. Constantinescu will also represent the Roumanian National Zoötechnical Institute.

Lt. Col. P. J. Simpson has been chosen to represent the Royal Counties Division of the National Veterinary Medical Association of Great Britain and Ireland.

Dr. B. Krumholz has been selected by the Palestine Veterinary Association as its official delegate.

Dr. E. H. Cook will do the honors for the Veterinary Medical Association of New Brunswick, Canada, at the Congress.

Maj. Gen. James J. B. Tapley will attend as the official delegate of the British Army Veterinary Services.

12th International Veterinary Congress
New York—August 13-18, 1934

CORYNEBACTERIUM INFECTION IN POULTRY*

By DIRAN M. YEGIAN, *Amherst, Mass.*
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On July 12, 1933, a Rhode Island Red pullet was submitted to the Diagnostic Laboratory. The bird had died within twelve hours. No history of previous sickness or of clinical symptoms was available. Postmortem examination showed emaciation and anemia. The liver was slightly enlarged. Petechial hemorrhages and numerous necrotic areas, 1 to 3 mm in size, were present. These necrotic areas were slightly elevated and irregularly circular. Direct smears made from liver, spleen, and necrotic areas showed numerous small, Gram-positive rods with parallel sides and somewhat rounded ends. The spleen also was enlarged, and minute necrotic areas were present. Excessive precardial fluid, percarditis and endocarditis were observed. A general hyperemic condition of the skeletal muscles and viscera was noted.

Cultural growth on meat extract agar inoculated from liver, spleen, pericardial fluid, and heart consisted of small, circular, convex, smooth, creamy-white colonies. They varied in diameter from 0.4 to 0.5 mm, and after incubation for 24 hours increased in size to a diameter of 0.5 to 1 mm. Incubation for over two weeks did not produce any change in the morphology and the character of the colonies. Thirty subcultures on different media did not show any variation. The colonies apparently were very consistent in character. These colonies could be pushed along the point of a needle, as if they were oily in character, but they emulsified readily. The growth in meat extract agar, chicken infusion agar, blood agar, and Loeffler's medium was not abundant. In dextrose medium growth occurred readily and abundantly. In broth, turbidity was produced without settling. Very little growth was observed in gelatin over an incubation period of five days. The organism apparently grows under aerobic conditions. The optimal temperature for growth was in the neighborhood of 37° C.

The organism was a short rod, 1 to 1.5 microns in length, with somewhat rounded ends and parallel sides. It was usually arranged in parallel rows, irregular groups, or sometimes occured singly. It stained homogeneously and was Gram-positive. There was no tendency to pleomorphism. Flagella and motility were absent. Capsulation did not occur. Spores were not formed. The older

*Contribution No. 187 from the Department of Veterinary Science, Massachusetts Agricultural Experiment Station. Received for publication, February 17, 1934.

cultures did not appear to show any changes in morphology and staining reaction. The organism produced acid, without gas, in dextrose, lactose, maltose, sucrose, dextrine, rhamnose, levulose, salicin and mannose. Slight acidity occurred in sorbite, arabinose, galactose, xylose, inulin, dulcite, amnite, inosite, and raffinose. These reactions were consistent over an incubation period of two weeks. Lactose and sucrose gave slight acidity in 24 hours and were strongly acid in 48 hours. Gelatin was not liquefied. Indol was not formed. Nitrates were not reduced. H_2S was not formed. Litmus milk was strongly acid, but no coagulation occurred.

Five 2-month-old birds were inoculated intraperitoneally with a saline suspension of the organism as shown in table I.

TABLE I—*Results of inoculation of birds with saline suspension of the organism.*

BIRD	APPROXIMATE DOSAGE (CC)	INOCULATED	NECROPSY	RESULTS
1	300	8–15	9–15	K. Organism not recovered
2	600		8–27	K. Organism recovered
3	600		9–25	K. Organism not recovered
4	900		8–21	D. Organism recovered
5	900		10–2	K. Organism recovered

K=Killed.
D=Died.

Postmortem examination of the bird which died revealed small necrotic areas and petechial hemorrhages on the liver. The spleen and heart appeared to be normal. An increased amount of pericardial fluid was observed. The organism was recovered from the liver, spleen, blood, and peritoneal cavity. Bird 1 did not show any clinical symptoms nor gross pathological changes. Bird 2 showed depression and anorexia during the third day after inoculation. On the fourth day, the bird appeared to be normal. Postmortem examination revealed four necrotic areas on the liver, 1 to 2 mm in size. The organism was recovered only from the liver. Bird 3 showed depression and was disinclined to move about on the third day after inoculation. These symptoms lasted for only 24 hours. The bird was somewhat emaciated. There were no gross pathological changes, and the organism was not recovered. Bird 5 showed depression and anorexia on the third day of inoculation. Although the bird recovered, it did not put on much weight. When necropsied, several necrotic areas were found on the liver and there was some evidence of chronic peritonitis.

The spleen and the heart did not reveal any gross pathological changes. The organism was recovered from the liver and the spleen.

The pathogenic action of the organism was tested in day-old chicks. Ten chicks were inoculated intraperitoneally, ten orally, and five were kept as controls. The inoculations were made with a saline suspension of the organism, equal to the turbidity of tube 2 of MacFarland's nephelometer. Each chick received 0.5 cc of suspension. Four chicks of the group inoculated intraperitoneally died within five days after the inoculation. The rest of the chicks showed clinical symptoms of the disease in various degrees. The symptoms lasted about two days. There was no mortality among the chicks which were inoculated orally. However, slight diarrhea was present in three chicks. The control chicks remained normal. The chicks which died revealed enlarged livers with pin-point necrotic foci. General hyperemia of the skeletal muscles and of the viscera was present. The organism was recovered from these four chicks from the heart, liver, spleen and lungs. The remaining chicks were destroyed and necropsied 21 days after the inoculations. The organism was not recovered from these chicks.

The pathogenic action of the organism was tested in guinea pigs. Six guinea pigs, weighing from 200 to 250 grams, were inoculated intraperitoneally with a saline suspension of the organism, equal to the turbidity of tube 2 of MacFarland's nephelometer. Table II shows the dosage and the summary of the results.

TABLE II—*Results of inoculation of guinea pigs with saline suspension of the organism.*

GUINEA PIG	DOSAGE (CC)	INOCULATED	NECROPSY	RESULTS
1	1.0		10–5	K. No clinical symptoms. Organism not recovered
2	1.5		9–30	K. Organism recovered
3	1.5	9–20	9–25	D. Organism recovered
4	2.0		10–25	D. Organism recovered
5	2.0		10–4	K. Organism not recovered
6	2.5		9–26	D. Organism recovered

K=Killed.
D=Died.

Manifestation of clinical symptoms and gross pathological changes in guinea pigs were comparable with those of the chickens. From the nature of his experiment, it is hard to draw conclusions on the pathogenic action of the organism. However, it seems that the organism may prove fatal to chickens and guinea pigs under favorable conditions.

To determine antigenic properties, two pullets were inoculated with a live saline suspension of the organism equal in turbidity to tube 3 of MacFarland's nephelometer. Each bird received 12 cc of the antigen intraperitoneally within ten days. The daily dosage was 2 cc for each bird. The birds were bled five days after the last inoculation. The highest agglutination titre obtained was 1:160. The antigen, when agglutinated, was cohesive rather than flocculent in character. The titre declined to 1:10 within four weeks of the last test.

While the foregoing characteristics of this organism suggest that it belongs to the genus Corynebacterium, no species of this genus has been described to which it corresponds. The differential characteristics of the eight species of Corynebacterium and the fermentation reactions of the eleven groups of diphtheroid bacilli tabulated by the Committee of the Medical Research Council were referred to in this study. At times, this organism may prove to be pathogenic, in large doses, to chickens and guinea pigs. The organism appeared to be slightly antigenic.

Wellcome Research Institution Exhibits Again

The exhibit of the Wellcome Research Institution, of London, England, at the 1934 Chicago International Exposition continues to be one of the chief attractions in the Hall of Science, as it was at A Century of Progress last summer.

One of the most striking features of the entire exhibit is the Historical Medical Museum, which illustrates the evolution and practice of medicine, surgery and allied sciences throughout the world from prehistoric times. Represented with interesting exhibits also are the Institution's Bureau of Scientific Research; Entomological Field Laboratories; Physiological Research Laboratories; Chemical Research Laboratories and the Museum of Medical Science.

Austria Fights Warble Fly

Austria has joined those countries that have made the treatment of cattle against the warble fly compulsory. Owners are required to destroy the larvae in the backs of animals before they are turned out to pasture. Failure to do this is punishable by a fine of approximately $100, or imprisonment for two months.

Veterinary Record.

THE STILLBORN PIG*

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The occurrence of young, which are born dead is not characteristic of swine alone but is a phenomenon which manifests itself rather widely among all mammals. Unfortunately, there are not readily available any extensive data upon which one can base an accurate opinion relative to the comparative frequency with which stillbirths occur in different species. This is perhaps more true of the domestic animals than it is of the human, since in the latter case the occurrence of a stillbirth becomes a matter of medical record. Among domestic animals, young which are dead at birth are usually disposed of without any particular observations having been made and frequently without the occurrence becoming a matter of record. As a result, the data on stillbirths among our common domestic animals are not only rather fragmentary but also widely scattered. It is the purpose of this short paper to analyze data pertaining to stillbirths in swine.

MATERIAL

The data which were available for study comprise a total of 497 pigs farrowed dead in 259 litters at the U. S. Animal Husbandry Experiment Farm, Beltsville, Maryland, during the period 1921-1932 inclusive. Among these pigs were represented several purebreds, a few grades and some crossbreds. Since preliminary studies indicated that breed did not have a decisive influence upon the frequency with which stillborn pigs appeared, the data for all breeds were combined.

As a preliminary step to the statistical analysis, the data were separated into three groups: the pigs born dead, live pigs born in litters containing also pigs born dead, and pigs in litters containing only live pigs. The data on sex ratio, size of litter and birth weight for these three groups are given in table I. Each of these items will be discussed separately in relation to the stillborn condition.

SIZE OF LITTER A FACTOR

The 8,991 pigs from which data were obtained for this study were farrowed in 1,121 litters, of which 259 contained one or more pigs dead at birth. Thus 23.10 per cent of all litters contained one or more stillborn young. The mean number of young

*Received for publication, February 26, 1934.

TABLE I—*Relation of sex to size of litter in litters having all young born alive and in litters which contain one or more pigs born dead.*

Litter Size	Pigs of Each Sex		Pigs Born Dead		Pigs Born Alive in Litters Containing Stillborns		Pigs in Litters Having All Pigs Born Alive	
	Females	Males	Females	Males	Females	Males	Females	Males
1	4	12	1	3	...	...	3	9
2	18	38	2	12	2	4	14	22
3	57	87	3	5	3	10	51	72
4	103	125	5	5	10	12	88	108
5	188	252	4	14	22	25	162	213
6	331	371	26	17	68	51	237	303
7	446	534	18	12	56	68	372	454
8	553	631	16	22	63	59	474	550
9	603	630	27	36	114	111	462	483
10	637	643	19	18	96	97	522	528
11	466	513	17	16	106	114	343	383
12	292	320	35	31	99	147	158	142
13	237	257	18	28	91	110	128	119
14	128	138	14	16	67	57	47	65
15	93	102	7	10	51	52	35	40
16	52	60	12	10	5	5	35	45
17	24	27	7	7	17	20	...	...
19	11	8	3	1	8	7	...	...
Totals	4,243	4,748	234	263	878	949	3,131	3,536

for all litters containing stillborns was 8.56, while for litters having all young born alive it was 7.73. There was, therefore, a tendency for litters containing stillborn pigs to be larger than litters containing only live pigs. This does not mean, however, that there was indicated a tendency for the number of stillbirths to increase uniformly with an increase in litter size. The graph in figure 1 shows clearly that this is not the case. Very large and very small litters contain a much higher percentage of stillbirths than do the litters of medium size. For instance, of 1,821 pigs farrowed in litters of one, two, twelve and above, 11.7 per

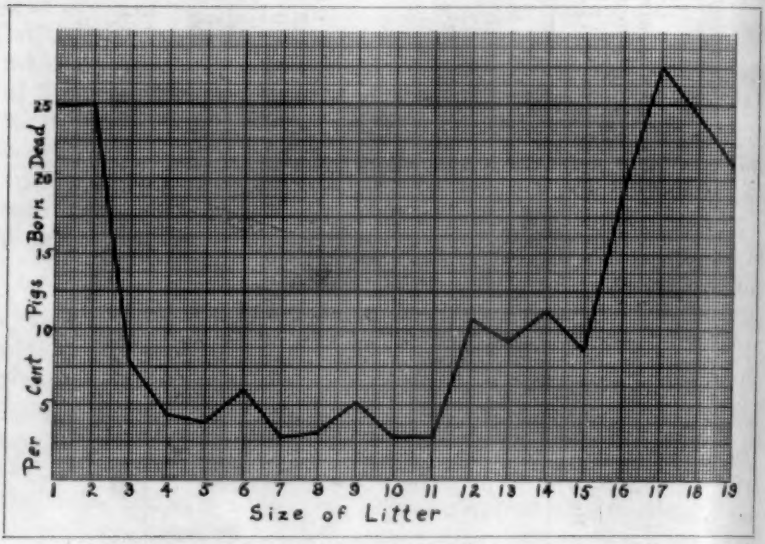

FIG. 1. The relation of litter size to percentage of stillbirths in a population of 8,991 pigs comprising 1,121 litters. The highest percentage of stillbirths occurs in the extreme litter sizes.

cent were dead at birth. Only 3.9 per cent of the 7,270 pigs farrowed in all the other litter sizes were dead at birth.

A high percentage of young born dead is not particularly surprising among very large litters and possibly can be accounted for on the assumption that such large litters make an excessively heavy physiological demand upon the mother. Such reasoning is hardly permissible, however, in the case of very small litters where the physiological demands of the litter must be far below the capacity of the dam. It seems more logical to assume that litters of one and two are really the remains of a much larger number, the major portion of which failed to develop to term. Naturally this brings us to a consideration of the destiny of the developing fetus during the gestation period.

A certain potential number of young are produced by a sow at each ovulation period. Whether the minute egg cells develop into young pigs which are born alive depends primarily upon their fertilization by sperm cells and only secondarily upon events which transpire subsequently. Size of litter at term is, of course, controlled in most cases not by the number of egg cells liberated by the ovaries but by the number of fertilized ova which survive. The studies of Corner[1] have shown that about 30 per cent of the fertilized egg cells fail to reach birth as pigs. This loss was distributed approximately equally among failure of eggs to segment, degeneration of the embryonic blastocyst and resorption between this point and birth. To this should be added the approximately 5.5 per cent which are born dead in order to arrive at an expression for the proportion of egg cells liberated which are actually farrowed as live pigs. Apparently this figure is about 66 per cent. In other words, at birth we are dealing with only about two-thirds of the egg cells which were liberated by the ovaries 114 days before.

Unfortunately the data in table I do not give any information at all relative to primary litter size, that is, the number of fertilized ova which were originally present in each case. Without such information it is impossible to ascertain whether there was greater percentage resorption in small litters than in large litters having about the same percentage of stillborn young.

Sex Ratio

The proportion of males among the 8,991 pigs included in this study is 52.23 ± 0.35. This figure approximates very closely similar data published by other workers and probably can be considered a good average for the species in general. The proportion of males shown by the three separate groups being considered in this study are as follows: born dead, 52.92 ± 1.51; live pigs in litters containing dead pigs, 51.94 ± 0.79; live pigs in litters containing only live pigs, 53.04 ± 0.41. The differences are in no case statistically significant and indicate that the stillborn condition in this group of pigs was not associated with sex. It was entirely a matter of chance whether males or females were born dead.

Birth Weight

Mean birth weight in the two groups of live pigs is about the same but that of the stillborn group is about one-half pound less. The unadjusted averages shown in table II for the two groups of live pigs probably differ significantly as they stand, but when adjustment for differences in distribution of litter size in the two

TABLE II—*Relation of mean birth weight to size of litter and sex among pigs born dead, pigs born alive in litters containing stillborns and in litters in which all pigs are born alive.*

Litter Size	Mean Birth Weight of Pigs Born Dead (Pounds)		Mean Birth Weight of Pigs Born Alive in Litters Containing Stillborns		Mean Birth Weight of Pigs Born in Litters Having All Pigs Born Alive	
	Females	Males	Females	Males	Females	Males
1	2.50	1.83	....	....	4.00	2.75
2	2.50	3.25	3.25	2.81	2.80	2.66
3	2.90	3.00	2.83	3.50	2.74	2.86
4	2.15	2.50	2.35	2.87	2.79	2.90
5	2.50	1.73	2.51	2.53	2.75	2.90
6	2.60	2.69	2.93	2.94	2.77	2.85
7	2.28	2.15	2.82	3.05	2.80	2.87
8	1.81	1.96	2.74	2.85	2.73	2.79
9	2.09	2.08	2.70	2.72	2.63	2.71
10	2.13	2.10	2.43	2.41	2.62	2.70
11	2.18	1.98	2.41	2.61	2.56	2.64
12	1.84	2.02	2.52	2.63	2.57	2.68
13	2.03	2.25	2.50	2.62	2.42	2.60
14	1.71	1.80	2.50	2.51	2.19	2.09
15	1.96	2.15	2.45	2.67	2.12	2.56
16	1.06	1.18	1.90	2.60	2.21	2.34
17	1.93	2.11	2.32	2.40	....	....
19	2.00	2.00	2.06	2.36	....	....
Total Average	2.05 ± 0.053	2.13 ± 0.057	2.57 ± 0.024	2.67 ± 0.024	2.65 ± 0.012	2.74 ± 0.012

groups is made, the averages, in pounds, become: all born alive, males 2.78, females 2.69; pigs born alive in litters containing stillbirths, males 2.76, and females 2.69. It is clear from these figures that the average birth weight of live pigs is not affected by the presence of stillbirths in the same litter.

The mean birth weights of the stillbirths were 2.13 pounds for the males and 2.05 for the females. The adjustment for differences in litter size changes these figures to 2.22 for the males and 2.14 for the females. The fact that these averages are statistically significant from average birth weight of both the groups of live pigs indicates strongly that the stillbirths as a group have for some reason grown at a slower rate or for a shorter period of time than did the pigs which were born alive.

The comparative variability of birth weight in the three groups throws some light upon this point. In litters having all young born alive the coefficient of variability was 25.7 for males and 23.8 for females. In the group of live pigs born in litters containing some stillbirths also the coefficient of variability for males was 27.2 and for females 27.4. In the stillborn group, however, the coefficients were 43.1 for males and 39.5 for females.

The much greater variability of birth weight in the stillborn group expresses statistically the same situation that observation of such pigs at parturition shows in a visual way; namely, that pigs born dead cover a range in size from the small, underdeveloped specimen to the large, fully developed fetus. It is possible that some of the fully developed individuals were born dead because of some accident at parturition. It is also likely that the underdeveloped specimens ceased growth several days prior to parturition and were in various stages of retrogression. The litters in which they appeared were not farrowed any earlier, however, than litters not having stillbirths. The mean gestation period for litters having one or more dead young was 115.05 ± 0.0900 and for litters in which all young were born alive it was 115.12 ± 0.0507. Of course, there still remains the probability that the young which are dead at birth died several hours or even days prior to parturition. Unfortunately, autopsy data, which might throw some light upon this point, are lacking.

The relationship between sex and birth weight seems to be independent of the factors affecting the stillborn condition. In all three groups the male pigs weighed more at birth than the females. The amounts by which the males exceeded the females in each group are as follows: stillborns, 0.08 pound; live pigs in litters containing young born dead, 0.07 pound; and live pigs in

litters containing only live pigs, 0.09 pound. McKenzie[2] found in his study of 60 sows and their progeny that male pigs exceeded females in birth weight by 0.09 pounds.

Discussion

It has been shown in this report that from 5 to 6 per cent of all pigs farrowed are dead at birth. It is not certain that this is a representative figure which would apply generally. Other investigators have reported slightly higher percentages. Carmichael and Rice[3] reported 7.5 per cent stillborn pigs among a large group, representing different breeds and ages of sows. McKenzie[2] reported as high as 10 per cent in certain experimental groups. It seems probable, therefore, that the estimate given in the present report is a very conservative one.

The loss due to stillbirths is not very large when considered in comparison with other prenatal and postnatal losses. Stillbirths constitute the visible portion, however, of a prenatal death loss which probably destroys as much as 35 per cent of all young which start in the beginning as fertilized eggs. There is possibility that the adjustment of conditions which result in the occurrence of stillbirths would also bring about the elimination of at least a portion of the other prenatal losses. And to this extent stillbirths may be even more important than their percentage occurrence might indicate.

The probable causal factors associated with the production of stillborn pigs are primarily biological in nature and are concerned with the physiology of prenatal development, the nutrition, age, and care of the mother and the occurrence of pathological conditions. Inheritance may be involved through the occurrence of gene complexes which are essentially lethal in effect. There is also the possibility that inheritance affecting the endocrine system may be involved. Critical study of all these phases of the situation is an urgent need in order that further results of practical value to the swine industry may be obtained. In the meantime the best that we can do is to apply more generally the knowledge which is available relative to the proper care and feeding of the sow both before and during gestation.

Summary

A study was made of pigs dead at birth in a group of 8,991 individuals farrowed in 1,121 litters at the U. S. Animal Husbandry Experiment Farm, Beltsville, Maryland.

The total number of young born dead was 497, which occurred in 259 litters.

Very large and very small litters contained a higher percentage of stillborn young than did the litters of medium size. Litters of one, two, twelve and above averaged 11.7 per cent dead at birth, whereas all other litters averaged 3.9 per cent.

Pigs dead at birth were significantly higher in weight than pigs born alive. There was no difference in the mean birth weight of live pigs farrowed in litters containing stillbirths and that of pigs farrowed in litters containing only live pigs.

The average birth weight of males exceeded that of females by about the same amount, regardless of whether the pigs were born dead or alive.

Birth weight was much more variable in the stillborn than in the live pigs.

A brief discussion is given of the probable causal factors concerned in the production of stillbirths and there is indicated a need for critical investigation of the biological problems involved.

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Motorists Attention

Members of the A. V. M. A., who are planning to motor to New York City to attend the Twelfth International Veterinary Congress and the A. V. M. A. meetings, August 13-18, may find it worth their while to get in touch with the Conoco Travel Bureau, Denver, Colo. The Bureau offers free travel service and will send on request a set of maps marked to show the shortest and best routes and the condition of the roads, illustrated literature descriptive of places of interest, lists of hotels and cottage camps, and a Conoco passport. Those interested in obtaining this service should write the Bureau, giving the approximate date of their departure and their destination.

Vermont Veterinarians to Meet in New York

There will be a business meeting of the Vermont Veterinary Medical Association at the Waldorf-Astoria Hotel, New York, Tuesday evening, August 14, at 5 o'clock, according to information supplied by Dr. G. N. Welch, Secretary. The room in which the meeting will be held will be announced on the bulletin boards at the Waldorf-Astoria.

STUDIES OF FIVE BRUCELLA ABORTUS (BOVINE) STRAINS AS IMMUNIZING AGENTS AGAINST BANG'S DISEASE (INFECTIOUS ABORTION)*

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INTRODUCTION

The determination of the most satisfactory degree of virulence for strains of *Brucella abortus* to possess for vaccine preparation, from the standpoints of both efficacy and safety, is a problem to which the writers have given much attention during the last few years. The results of experiments dealing with the subject have already been published.[1-3] The most promising results, everything considered, were obtained from the use of strains of the bovine type so low in virulence as only occasionally to cause slight lesions in guinea pigs which received, subcutaneously, 0.25-cc. doses of suspensions ten times the density of tube 1 of the McFarland nephelometer but induced agglutinins in their blood in titres of from 1:50 to 1:500 when killed two months later. *Br. abortus* strains of this degree of virulence gave evidence of inducing a distinct immunity when administered subcutaneously to calves, virgin heifers and open cows, and of being at the same time practically free from the most objectionable features which result from the use of virulent cultures.

Br. abortus strains of sufficient virulence to cause lesions in guinea pigs regularly when injected in the same amounts and in like manner were found often to localize in the udders of the vaccinated cows and to be eliminated in their milk, a feature that renders such strains seriously objectionable for vaccine preparation, even though they were shown to be immunity-producing for the virgin heifers, calves and unbred cows in which they did not become localized.

The use of avirulent strains of *Br. abortus* eliminated this objectionable feature, but there was some evidence which indicated that such strains lose much of their immunizing value with the disappearance of their pathogenicity. An experiment was inaugurated accordingly to throw more light on this matter. While the results of this experiment in a measure confirmed those previously obtained, it still seemed desirable to carry the investigation further. The results of the later work plainly indicated that the avirulent *Br. abortus* strain employed possessed

*Received for publication, May 19, 1934.

little, if any, virtue as an immunizing agent when administered to virgin heifers of near breeding age, whereas a strain of low virulence gave evidence of conferring marked resistance to the disease. Inasmuch as these two experiments are much alike and some of the same strains of *Br. abortus* were used in both, they are discussed together in this paper as vaccine experiments 4 and 5.

Vaccine Experiment 4

In vaccine experiment 4, efforts were made to test the relative efficacy of four *Br. abortus* (bovine) strains as immunizing agents for open cows and virgin heifers of near breeding age. Two of the strains (801 and 802) were avirulent for guinea pigs and the other two strains (11 and 19) were of low virulence but not entirely avirulent for these animals.

Vaccines Used and Tests for Virulence

Vaccines were prepared from 72-hour growths of each of the strains and adjusted to a density ten times that of tube 1 of the McFarland nephelometer. The vaccines were administered subcutaneously in 10-cc doses to 16 virgin heifers and three open cows, all of which were negative to the abortion agglutination test. Four heifers received vaccine prepared from strain 801; four heifers, vaccine prepared from strain 802; four heifers and one cow, vaccine prepared from strain 11, and four heifers and two cows vaccine prepared from strain 19. Eight virgin heifers negative to the agglutination test were reserved for controls.

To determine the effects of the four vaccines on guinea pigs, twelve were injected subcutaneously with each one, each guinea pig receiving 0.25 cc. The guinea pigs were killed two months later and the blood serum of each was subjected to the agglutination test. Average agglutination titres, based on dilutions of 1:25, 1:50, 1:100, 1:200 and 1:500, were determined for each group.

The vaccine prepared from strain 801 produced no lesions and the average agglutination titre was 1:162. The vaccine prepared from strain 802 produced no lesions and the average agglutination titre for the group was 1:77. The vaccine prepared from strain 11 produced slight spleen lesions in four of the twelve guinea pigs inoculated with it and an average agglutination titre of 1:275, and the vaccine made from strain 19 produced slight spleen lesions in two of the twelve guinea pigs inoculated with it and an average agglutination titre of 1:408.

Temperatures of the cattle were taken just before vaccination on May 19, 1930, and on the first five days following. The tem-

peratures were commonly from two to five degrees above normal on the first day after vaccination. The cattle which had received the vaccines of low virulence showed slightly more elevated temperatures than those which received the avirulent vaccines. The temperatures usually remained somewhat above normal for three days, after which they rapidly subsided. Nearly all of the vaccinated animals showed slight swellings at the sites of vaccination which persisted for four or five days and then gradually disappeared. One of the vaccinated animals refused feed for a day or two, but the appetites of the remainder appeared to be unaffected.

The principals were tested repeatedly for the presence of *Br. abortus* agglutinins, at intervals of from two to five weeks throughout the experiment. Although the animals were negative to the agglutination test at time of vaccination, all reacted in high titres 15 days later. The average titre of those which received the vaccines of low virulence was at this time 1:1,772 and that of those which received the avirulent vaccines 1:550. Four and one-half months after vaccination, six of the eight heifers which received the avirulent vaccines gave negative results to the agglutination test, the remaining two reacting in a titre of 1:50 only. At the same time the average titre of the animals which received the vaccines of low virulence was 1:233 and none of the animals reacted in a titre lower than 1:100.

Breeding of the cattle was commenced early in July, 1930, about seven weeks after they were vaccinated, and was continued through August and September, use being made of a bull negative to the agglutination test. At the commencement of breeding, the average agglutination titre of the animals which received the avirulent vaccines was 1:75 and the average titre of those which received the vaccines of low virulence was 1:400.

On October 16, 1930, a little less than five months after vaccination, the cattle were examined for pregnancy. The vaccinated group now consisted of but 17 animals, two of those which received the vaccine prepared from strain 11 having died as a result of consuming grass on which a small amount of lead paint had been sprayed accidently. Thirteen of the 17 vaccinated cattle were found to be pregnant. Pregnancy could be recognized, however, in only two of the eight heifers which had been reserved for controls, although efforts had been made to breed them during the same period that the principals were being bred. A number of other pregnant heifers, negative to the agglutination test, were nevertheless available and these were substituted for the controls that failed to conceive. The average agglutina-

tion titre of the pregnant animals which received the avirulent vaccines was now 1:45 and those which received the vaccines of low virulence, 1:287.

Exposure of Cattle to Brucella Abortus

On October 22, 1930, 13 principals and seven controls were given exposure by depositing, on the conjunctiva of one eye of each animal, three drops of a suspension of *Br. abortus* 20 times the density of tube 1 of the McFarland nephelometer. The suspension was prepared from 48-hour growths of *Br. abortus* strain 613, the second transfer, and was shown to have pronounced virulence for guinea pigs.

On December 13, 1930, the four remaining principals and one control, which upon examination were found to be pregnant, received *Br. abortus* conjunctival exposure. The *Br. abortus* suspension used at this time for making the exposure was 20 times the density of tube 1 of the McFarland nephelometer and was prepared from *Br. abortus* strain 1496, the tenth transfer. The suspension was shown to be decidedly virulent for guinea pigs. As in the previous exposure, three drops of the suspension were deposited on the conjunctiva of one eye.

Records of the cattle were kept, showing whether they produced weak or vigorous calves or aborted, and six guinea pigs were injected with uterine material and the same number each with 5 cc of a composite sample of colostrum of each animal at time of calving or aborting.

Results

Of the four heifers which received the avirulent vaccine prepared from strain 801, three produced vigorous calves and one aborted. *Br. abortus* was proved to be present in the uterine material and colostrum of the heifer which aborted and in the colostrum of two of the three heifers which produced vigorous calves.

Of the four heifers which received the avirulent vaccine prepared from strain 802, three produced vigorous calves and one aborted. *Br. abortus* was proved to be present in the uterine material and colostrum of the heifer which aborted and in the uterine material and colostrum also of one of the three heifers which produced vigorous calves. Results of guinea-pig inoculations with the uterine exudates and colostrum of the other two heifers were negative.

Of the three surviving animals of the group which received a vaccine of low virulence prepared from strain 11, all produced vigorous calves, but *Br. abortus* was shown to be present in the

uterine material and colostrum of one and in the colostrum of another. Results of guinea-pig inoculations with uterine material and colostrum of one heifer and the uterine exudate of another were negative.

Of the six animals which received the vaccine of low virulence prepared from strain 19, four produced vigorous calves and two aborted. *Br. abortus* was proved to be present in the uterine material and colostrum of one of the heifers which aborted but the absence of the infection in both of these substances was indicated by the inoculation results in connection with the other aborter, as also with the four animals which produced vigorous calves.

Of the eight controls, all aborted and *Br. abortus* was shown to be present in the uterine exudates and colostrum of all of them.

Table I shows the date of vaccination, the dates of breeding and of *Br. abortus* exposure, how the gestation periods terminated and the results of guinea-pig inoculations with uterine fluids and colostrum.

Discussion of Results

The *Br. abortus* exposure to which the animals were subjected in this experiment was very severe, as is indicated by the fact that all of the controls acquired the disease and aborted. Yet, under these conditions only one of the six animals which received the vaccine prepared from *Br. abortus* strain 19 gave evidence of having become infected with the disease as a result of the exposure. Of the nine animals which received the vaccines prepared from the strains of low virulence (11 and 19), 33.3 per cent were shown to be affected with the disease at time of calving or aborting, while of the eight animals which received the avirulent vaccines (801 and 802), 62.5 per cent were shown to be infected with *Br. abortus* at this time, although 75 per cent gave birth to vigorous calves.

The results of this experiment, while indicating that two avirulent strains, when used in connection with unbred cattle, possessed some virtue as immunizing agents, showed that they were much less efficient than two other strains which still possessed slight virulence. The two avirulent strains used (801 and 802) seemed to differ but little from each other in efficacy from an immunizing standpoint.

Vaccine Experiment 5

In vaccine experiment 5, consideration again was given to the matter of virulence of *Br. abortus* strains as immunizing

agents for infectious abortion. Use was made in this experiment of one of the avirulent *Br. abortus* (bovine) strains (801) that was used in vaccine experiment 4, a single strain of low virulence (19) also used in vaccine experiment 4, and one strain (618) that regularly produced lesions in guinea pigs.

Thirty-seven heifers of near breeding age and negative to the agglutination test for Bang's disease were used in this experiment.

Vaccines Used and Tests for Virulence

Vaccines were prepared from 72-hour growths of each of the three strains (801, 19 and 618) and adjusted to a density ten times that of tube 1 of the McFarland nephelometer. To test the virulence of the vaccines, 18 guinea pigs were injected subcutaneously with 0.25 cc of each of the vaccines and killed at the end of two months, when samples of their blood serum were subjected to the agglutination test. None of the guinea pigs which were injected with the vaccine prepared from strain 801 showed lesions and all except one gave negative results to the *Br. abortus* agglutination test. This pig reacted in a titre of 1:25. Four of the 18 guinea pigs which were injected with the vaccine prepared from strain 19 showed slight spleen lesions of abortion disease but in the fourteen other guinea pigs abortion-disease lesions were absent. The average agglutionation titre of this group of guinea pigs, based upon the titres obtained in serum dilutions of 1:25, 1:50, 1:100, 1:200 and 1:500, was 1:304. All of the guinea pigs injected with the vaccine prepared from strain 618 showed extensive abortion-disease lesions and had an average agglutination titre of 1:500, based upon the five serum dilutions mentioned.

On February 15, 1932, eleven heifers were injected subcutaneously with vaccine prepared from the avirulent strain, 801; nine with vaccine prepared from strain 19; and six, with that prepared from strain 618. Eleven heifers were reserved for controls. About half the principals received 10-cc and the other half 20-cc doses.

Most of the vaccinated cattle had temperatures from two to four degrees above normal for three days following the vaccine injections. The thermal reactions were somewhat less marked in the cattle which received the avirulent vaccine than in those which received either of the other two.

Nearly all of the animals showed slight swellings at sites of vaccination, which persisted for about a week. Several of the heifers were off feed the day after they were vaccinated but their appetites soon returned.

TABLE I—*Immunizing results with cattle injected with vaccines prepared from four Brucella abortus strains, the animals later being subjected to artificial Br. abortus conjunctival exposure.*

ANIMAL	DATE OF VACCINATION (1930)	VACCINE	DATE OF BREEDING (1930)	DATE OF EXPOSURE (1930)	GESTATION PERIOD (DAYS)	OUTCOME OF PREGNANCY	RESULTS OF EXAMINATIONS FOR BR. ABORTUS	
							UTERUS	COLOSTRUM
H1595	May 19	Strain 801 avirulent	0	Dec. 13		Abortion	+	+
H1600			0	Dec. 13		Vigorous calf	−	−
H1601			Aug. 13	Oct. 22	285	Vigorous calf	−	+
H1626			Sept. 13	Oct. 22	275	Vigorous calf	−	+
H1599	May 19	Strain 802 avirulent	July 26	Oct. 22	283	Vigorous calf	−	−
H1603			Aug. 21	Dec. 13	226	Abortion	+	+
H1612			Aug. 23	Oct. 22	285	Vigorous calf	−	−
H1623			Aug. 20	Oct. 22	286	Vigorous calf	+	+
H1596	May 19	Strain 11 low virulence	Aug. 27	Oct. 22	282	Vigorous calf	+	+
H1598			July 9					
			Sept. 3	Oct. 22	286	Vigorous calf	−	−
C1609			Aug. 23	Oct. 22	282	Vigorous calf	−	+

TABLE I—*Immunizing results with cattle injected with vaccines prepared from four Brucella abortus strains, the animals later being subjected to artificial Br. abortus conjunctival exposure—Continued.*

ANIMAL	DATE OF VACCINATION (1930)	VACCINE	DATE OF BREEDING (1930)	DATE OF EXPOSURE (1930)	GESTATION PERIOD (DAYS)	OUTCOME OF PREGNANCY	RESULTS OF EXAMINATIONS FOR BR. ABORTUS	
							UTERUS	COLOSTRUM
H1597	May 19	Strain 19 low virulence	0	Oct. 22		Abortion	−	−
H1616			Sept. 20	Dec. 13	281	Vigorous calf	−	−
H1624			Aug. 7	Oct. 22	281	Vigorous calf	−	−
H1628			July 7	Oct. 22	196	Abortion	+	+
C1551			July 29	Oct. 22	286	Vigorous calf	−	−
C1552			Sept. 22	Oct. 22	287	Vigorous calf	−	−
H1430	Controls		0	Oct. 22		Abortion	+	+
H1432			0	Oct. 22		Abortion	+	+
H1480			0	Dec. 13		Abortion	+	+
H1481			0	Oct. 22		Abortion	+	+
H1550			0	Oct. 22		Abortion	+	+
H1577			July 10	Oct. 22	197	Abortion	+	+
H1584			July 6 Sept. 9	Oct. 22	126	Abortion	+	+
H1589			0	Oct. 22		Abortion	+	+

Key: O = No record. + = Presence of *Br. abortus*. — = Absence of *Br. abortus*. H = Heifer. C = Cow.

Repeated agglutination tests were made of the blood serum of the vaccinated heifers at frequent intervals. Ten days following the injections, the average titre of the heifers vaccinated with strain 801 was 1:700; strain 19, 1:1,132; and strain 618, 1:1,200. On May 4, 1932, approximately three months following vaccination, the average agglutination titre of the heifers vaccinated with strain 801 was 1:36; with strain 19, 1:177; and with strain 618, 1:164.

Breeding of the animals was commenced early in May and continued through June, 1932, a bull negative to the abortion agglutination test being used. They came in heat promptly and a large portion of them were bred during the first month.

Examination of the heifers for pregnancy was made August 29, 1932, when 34 of the 37 were found to be pregnant. Ten of the 34 had received strain 801 vaccine; eight, strain 19 vaccine, and six, strain 618 vaccine. Ten were unvaccinated controls. The three heifers in which pregnancy was not demonstrated were found afterwards to be in calf and were exposed at a later date. The four groups were given conjunctival exposure the following day.

Exposure of Cattle to Brucella Abortus

Results from the use of the conjunctival method of exposure in previous vaccine experiments had suggested that this method is unusually severe, since all or nearly all of the controls treated by it commonly acquired the disease. It was proposed, therefore, in this experiment to reduce the severity of the exposure somewhat with the view to more nearly approaching the natural exposure that prevails in herds in which the disease is active.

Br. abortus strain 1531 had recently been used to give conjunctival exposure in another vaccine experiment. This strain had been under artificial cultivation for nearly a year but, nevertheless, seemed to retain its original virulence for both guinea pigs and cattle and produced marked abortion-disease lesions in the former. It had appeared to furnish the desired exposure when applied to the conjunctiva in the experiment in which it had been used a few months earlier. As it was thought the strain by this time might have lost some of its virulence through cultivation on an artificial medium, use was made at this time of an isolation of the strain from a heifer caused to abort by exposure to the original strain. It was believed that as a result of its sojourn in the heifer its virulence had been somewhat increased. Three drops of a suspension twice the density of tube 1 of the McFarland nephelometer, prepared from 72-hour growths

of the strain, were deposited on the conjunctiva of one eye of each of the 34 animals in the experiment that were found to be pregnant, guinea pigs being injected subcutaneously with 0.25 cc doses of the suspension at the same time, as controls on its virulence. When the guinea pigs were killed, two months later, they all reacted strongly to the agglutination test but, contrary to expectations, they had developed only slight abortion-disease lesions. It was feared, therefore, that the *Br. abortus* exposure of the cattle had not been sufficiently severe. This, however, was proved not to be the case, as a high percentage of the control cattle acquired the disease and aborted.

Two vaccinated heifers and one control, in which pregnancy was not demonstrated when the first lot of 34 was exposed, were found later to be in calf and received conjunctival exposure, November 5, 1932. Three drops of a suspension prepared from *Br. abortus* strain 695, the eighth transfer, a different strain from the one used in giving exposure to the 34 heifers, adjusted to a density of tube 1 of the McFarland nephelometer, were deposited on the conjunctiva of one eye of each of these three heifers. This suspension regularly produced marked lesions of abortion disease in the guinea pigs which received 0.25 cc subcutaneously.

The average agglutination titre of the blood serum of the ten control heifers that were negative to the agglutination test when exposed August 30, 1932, was 1:247 a month later. The 24 principals which were exposed on the same date had an average agglutination titre of 1:220 a month afterward although, when exposed, their average titre was 1:99.

As in vaccine experiment 4, records were kept of the cattle, showing whether they produced vigorous or weak calves or aborted, and six guinea pigs were injected with uterine fluids and the same number with 5 cc of colostrum of each animal at time of calving or aborting.

Results

Of the eleven heifers which were vaccinated with the avirulent *Br. abortus* strain 801, eight aborted and three produced vigorous calves. The presence of *Br. abortus* was demonstrated in the uteri and colostrum of all eight heifers which aborted and in the uterus and colostrum of one heifer which produced a vigorous calf, but efforts, through the inoculation of guinea pigs, to isolate the infection from uterine material and colostrum of the remaining two heifers which produced vigorous calves, failed.

Of the nine heifers which were vaccinated with the strain of low virulence (19), six produced vigorous calves. Results of

guinea-pig inoculations indicated the absence of *Br. abortus* from their uteri and colostrum. Two heifers vaccinated with this strain aborted but from undetermined causes. Guinea pigs which were injected with uterine material and colostrum from each animal failed to acquire *Br. abortus* infection. The remaining heifer was unable to give birth to her calf because of its huge size and both dam and calf died. Results of guinea-pig inoculations with uterine material and colostrum were negative for *Br. abortus.*

Of the six heifers which were vaccinated with strain 618, all produced vigorous calves. Results of guinea-pig inoculation showed *Br. abortus* was present in the colostrum of one animal but not in the uterus and indicated the absence of the infection in both uterus and colostrum of each of the five other animals.

Of the eleven controls, seven aborted and four produced vigorous calves. *Br. abortus* was found to be present in the uterus of all seven of the aborters and in the colostrum of six. Efforts to isolate *Br. abortus* from the uteri and colostrum of the four heifers which produced vigorous calves failed.

Table II shows the date of vaccination, breeding dates, dose of vaccine administered, date of *Br. abortus* exposure, how gestation periods terminated and inoculation results derived from uterine materials and colostrum in connection with the animals in this experiment.

Discussion of Results

The results of experiment 5 indicate that the avirulent strain 801, in the 21 months that elapsed between the beginning of the fourth and fifth experiments, had lost the rather weak immunizing power it was shown to possess by experiment 4, since a higher percentage of the heifers vaccinated with it in the fifth experiment acquired the disease on exposure than of the unvaccinated controls. The strain also had changed in its agglutinin-inducing effect; for, when used in the earlier experiment, guinea pigs injected with vaccine prepared from it reacted with an average agglutination titre of 1:162 two months later. In the latter experiment all of the guinea pigs injected with like doses of vaccine prepared from the strain at this time failed to react at the end of two months except one and that reacted in a titre of but 1:25. It, therefore, seems reasonable to believe that the immunizing value of *Br. abortus* may be greatly reduced or entirely lost if, through long cultivation on artificial media, it has become so altered as no longer to produce lesions in guinea pigs and its agglutinin-inducing power much lowered or possibly lost.

The results obtained, as measured by bacteriological examination, from the use of *Br. abortus* strain 19 indicated that this strain was not only highly effective as an immunizing agent but immunity was induced without more than transient infection, since efforts to determine the presence of *Br. abortus* in uterine exudates and colostrum of the heifers which received this strain, at the termination of their gestation periods, failed. It seemed reasonable to conclude that the two animals, which aborted following vaccination with strain 19, were caused to abort by some factor or factors other than *Br. abortus*. While strain 19 was nearly non-lesion-producing for guinea pigs when used in this experiment, it differed considerably from strain 801 in that it caused guinea pigs to react to the agglutination test in decidedly higher titres when killed two months subsequent to their injection.

Although strain 19 was only to a slight degree lesion-producing for guinea pigs, it appeared to be quite as efficient as an immunizing agent as strain 618, which produced marked lesions in guinea pigs and caused them to react to the agglutination test in titres of 1:500 or higher when killed two months subsequently.

While *Br. abortus* strain 618 was used in this experiment with the view to ascertaining if a direct relation exists between the virulence of strains and their immunity-inducing power, strains as virulent as No. 618 have been shown to be seriously objectionable for immunizing purposes, since they are likely to become implanted in the udders of vaccinated cattle, especially open cows with functioning udders. Strain 19, on the other hand, was so low in virulence as practically to exclude this objectionable feature. Nevertheless, it seemed to be nearly or quite as effective in inducing immunity as a more virulent one.

SUMMARY

The results of two vaccine experiments are reported in which use was made of two avirulent *Br. abortus* (bovine) strains, two strains of low virulence and one of high virulence as immunizing agents.

In the first experiment the immunizing value of two avirulent strains and two strains of low virulence received consideration. The vaccines were used in connection with virgin heifers of near breeding age and open cows. During pregnancy the vaccinated animals with controls were subjected to *Br. abortus* conjunctival exposure. In this experiment the two avirulent strains gave evidence of possessing some immunizing value, since but 25 per cent of the animals vaccinated with them aborted and 37.5 per cent

TABLE II—*Immunizing results with heifers injected with vaccines prepared from three Brucella abortus strains, the animals later being subjected to artificial Br. abortus conjunctival exposure.*

HEIFER	DATE OF VACCINATION (1932)	VACCINE	DOSE (CC)	DATE OF BREEDING (1932)	DATE OF EXPOSURE (1932)	GESTATION PERIOD (DAYS)	OUTCOME OF PREGNANCY	RESULTS OF EXAMINATIONS FOR BR. ABORTUS	
								UTERUS	COLOSTRUM
1606			20	May 23	Aug. 30	209	Abortion	+	+
1700			20	May 17	Aug. 30	286	Abortion	+	+
1703			20	May 23	Aug. 30	158	Abortion	+	+
1704			10	May 20	Aug. 30	201	Abortion	+	+
1706			10	May 11	Aug. 30	230	Abortion	+	+
1707	Feb. 15	Strain 801	20	May 6	Aug. 30	234	Abortion	+	+
1713		avirulent	10	June 6	Aug. 30	191	Abortion	+	+
1716			10	May 11	Aug. 30	222	Abortion	+	+
1741			20	May 11	Aug. 30	286	Vigorous calf	−	−
1751			10	May 12	Aug. 30	286	Vigorous calf	−	−
1702			10	May 17 June 4	Nov. 5	268	Vigorous calf	+	+
1714			20	0	Aug. 30		Abortion	−	−
1717			10	May 26	Aug. 30	284	Vigorous calf	−	−
1730			10	May 5	Aug. 30	276	Vigorous calf	−	−
1735			10	May 15	Aug. 30	277	Dead calf (dystocia)	−	−
1737	Feb. 15	Strain 19 low virulence	20	May 11	Aug. 30	276	Vigorous calf	−	−
1738			20	0	Aug. 30		Abortion	−	−
1740			10	May 10	Aug. 30	278	Vigorous calf	−	−
1748			20	May 6	Aug. 30	278	Vigorous calf	−	−
1744			10	May 12 July 25	Nov. 5	278	Vigorous calf	−	−

TABLE II—*Immunizing results with heifers injected with vaccines prepared from three Brucella abortus strains, the animals later being subjected to artificial Br. abortus conjunctival exposure—Continued.*

HEIFER	DATE OF VACCINATION (1932)	VACCINE	DOSE (CC)	DATE OF BREEDING (1932)	DATE OF EXPOSURE (1932)	GESTATION PERIOD (DAYS)	OUTCOME OF PREGNANCY	RESULTS OF EXAMINATIONS FOR BR. ABORTUS	
								UTERUS	COLOSTRUM
1709	Feb. 15	Strain 618 high virulence	10	May 7					
				May 28	Aug. 30	279	Vigorous calf	−	±
1711			20	May 9					
				June 3	Aug. 30	280	Vigorous calf	−	−
1718			20	May 21	Aug. 30	285	Vigorous calf	−	−
1719			20	May 11	Aug. 30	280	Vigorous calf	−	−
1734			10	May 10	Aug. 30	280	Vigorous calf	−	−
1742			10	May 18	Aug. 30	289	Vigorous calf	−	−
1720	Controls			May 16	Aug. 30	223	Abortion	+	+
1724				May 11					
				May 20	Aug. 30	158	Abortion	+	+
1726				May 5	Aug. 30	235	Abortion	+	−
1732				May 5	Aug. 30	281	Vigorous calf	−	−
1736				May 12	Aug. 30	281	Vigorous calf	−	−
1743				May 23	Aug. 30	190	Abortion	+	+
1746				May 11					
				May 22	Aug. 30	199	Abortion	+	+
1747				May 6					
				May 14	Aug. 30	248	Abortion	+	+
1749				0	Aug. 30		Vigorous calf	−	−
1752				May 7					
				May 13	Aug. 30	286	Vigorous calf	−	−
1754				May 16					
				June 12	Nov. 5	237	Abortion	+	+

Key: 0 = No record. + = Presence of *Br. abortus*. − = Absence of *Br. abortus*.

resisted the disease following *Br. abortus* exposure that caused 100 per cent of the controls to acquire the disease and abort.

The two strains of low virulence were more effective as immunizing agents, however, since of the animals vaccinated with them, 66.6 per cent resisted the disease and only 11.1 per cent aborted as a result of *Br. abortus* infection.

In the second experiment the immunizing value of three strains, one virulent, one of low virulence and one of high virulence, was studied. The vaccines were used in connection with virgin heifers of near breeding age. As in the first experiment, exposure was made to *Br. abortus* during pregnancy, by the way of the conjunctiva.

The results indicate that the avirulent strain which gave evidence of considerably increasing resistance to infection in the first of the two experiments was without virtue as an immunizing agent in this one, since 81.8 per cent of the animals vaccinated with it acquired the disease following exposure that transmitted the disease to 63.6 per cent of the unvaccinated controls. Of the animals vaccinated with the strain of low virulence, 100 per cent resisted the disease, and of those vaccinated with the strain of high virulence, 83.3 per cent resisted it. When used in the second experiment, the avirulent strain (801) had been under cultivation 21 months longer than when used in the first experiment and apparently the last vestige of its immunity-inducing power had disappeared.

CONCLUSIONS

The data yielded by these experiments seem to justify the following conclusions:

It is possible for a strain of *Brucella abortus* to become so altered by continued artificial propagation that its immunizing value for cattle, as well as its lesion-producing power for guinea pigs, disappears, although it still retains, in a measure, its agglutinin-inducing power.

A strain of *Br. abortus*, so reduced in virulence that it caused slight spleen lesions only occasionally in guinea pigs, but continued to retain much of its original power of stimulating the production of agglutinins, gave evidence of conferring a distinct immunity to cattle.

The degree of immunity, conferred by a *Br. abortus* strain so low in virulence as to be practically free from danger when used in the preparation of vaccine to be administered to unbred cattle, appeared quite as pronounced as that induced by a much more

virulent strain, the use of which for vaccine preparation has serious objections.

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Notice of Examination for Appointment in the Veterinary Corps, Regular Army

Examination for the purpose of qualifying candidates for appointment in the Veterinary Corps, Regular Army to fill eight existing vacancies, will be held October 8 to 13, 1934, inclusive.

Application blanks, Form No. 62, A.G.O., may be obtained from The Adjutant General or The Surgeon General, U. S. Army, Washington, D. C., or from the Commanding Officer or Surgeon of any military post or station, and when completed should be forwarded direct to The Adjutant General, U. S. Army, Washington, D. C.

The applicant must be a male citizen of the United States between the ages of 21 and 29 9/12 years, and a graduate of a recognized veterinary college.

An Invitation

The Pennsylvania State College, at State College, Pennsylvania, extends to the delegates to the Twelfth International Veterinary Congress and members of the American Veterinary Medical Association a cordial invitation to visit the campus of this College following the meeting in New York.

The calorimeter for large animals, the college herds and flocks, and the projects in relation to feeds and feedings are of special interest to veterinarians.

All-Breed Dog Show at Clarksdale

The North Mississippi Kennel Club, Clarksdale, will hold its first all-breed show on September 19-20, 1934, according to information received from Dr. Wm. L. Gates, chairman of the Bench Show Committee. Veterinarians who may wish to make entries in the show should get in touch with Dr. Gates, Box 417, Clarksdale, Miss. He will be pleased to supply the program and trophy list.

12th International Veterinary Congress
New York—August 13-18, 1934

LOOKING FORWARD WITH THE VETERINARY PROFESSION*

By LESLIE E. PIKE, *Long Beach, Calif.*

In the present historic days of world-wide readjustment, we are brought face to face with the problem of what the veterinary profession's sphere of usefulness is to be in the future. We know what has happened in the past. We have seen our members develop from a few self-educated, poorly equipped individuals to a body of highly trained professional men—men who are equipped with all the modern scientific means, that have been developed by years of research to alleviate and control the diseases of lower animals.

While we have received much, we also like to feel that we have contributed our share of general knowledge relative to the control of disease. I do not wish to indulge in an argument as to "who won the war," but it seems only fair to point out again that the work of the veterinary profession on vector diseases was the start of a chain of research that has brought relief to untold millions of people. That serum therapy, developed by veterinarians, is now being used extensively in human medicine, and that parasitological studies, made by brilliant members of our profession, have been of untold value in the relief of human suffering. The eradication of bovine tuberculosis and of glanders and the bringing of anthrax under control have been of benefit to public health as well as of economic importance. The constant war by the veterinary profession on "animal diseases akin to man" is little recognized by the general public. As sanitary officers, as inspectors of milk, as guardians of the meat-food supply of nations, veterinarians have made their mark.

I briefly mention some of the accomplishments of our profession for three reasons:

First, the only yardstick we have to measure future events is by what has happened in the past.

Second, it is only by impressing the public with what has been accomplished that we can hope to receive proper recognition and legislative protection in the future.

Third, we need to talk over our common heritage once in a while to bolster up our morale and revive our enthusiasm for greater efforts in the future.

*Presidential address delivered at the annual meeting of the California State Veterinary Medical Association, Berkeley, June 19, 1934.

This is the time for "looking forward." We have seen our profession change from what was originally a study of the equine species to a broad field of endeavor. Our knowledge now must embrace the entire realm of animal life. The birds of the air, the beasts of the field, and the fishes that live below the surface of the water—all come in for our attention.

It is now time to consolidate our gains, to plan for an even greater usefulness in the future. In view of our increased usefulness, why should we not receive greater recognition? Should we drift along as a partly organized class or should we stimulate the greatest minds of our profession to lay plans for advancement? We must not continue to allow the public to give us little consideration as learned men. We should unite a campaign to educate the public to know that when they consult a veterinarian, they are taking a proper step to protect their animals and themselves from loss through disease.

Stronger National Association Needed

First, I believe we need a more united and stronger national veterinary association, devoted not only to the dissemination of scientific knowledge, but to the economic welfare of its members as well.

No great movement affecting a large number of people was ever accomplished without organization. In the United States, we have developed a national association of veterinarians. Then we have state associations, such as this body meeting here today and there are also a large number of local groups that have banded themselves together into local associations.

There exists in this state, and I am sure also in other states, a certain percentage of veterinarians who probably consider themselves as belonging to the "rugged individualistic" class, who elect to go their own way, belonging to no association and doing practically nothing for the benefit of the profession. They perhaps consider that they owe nothing to organized veterinary medicine, but they forget that what protection they receive from veterinary practice acts is due to the efforts of association members who have secured the passage of such acts. They ignore the fact that the new knowledge they receive in the way of diagnosis and treatment is largely developed and presented through various associations. They fail to comprehend that in this day of organization in all lines of human endeavor, an unorganized veterinary profession would soon fall by the wayside.

I admit that veterinary practice acts are not perfect—that we have to meet unfair lay competition—that there are many things that need correction. With the united support of all members of our profession we could go a long way in correcting the abuses and unsatisfactory conditions that exist at the present time.

I have here some interesting figures that carry out my contention that veterinarians are not well organized. The 1930 census report gives the total number of veterinarians in the United States as 11,585. The A. V. M. A. membership for that year is given as 4,234. This indicates that we have only a trifle over one-third enrollment of veterinarians into the only association that is large enough to do things on a national scale.

I do not know whom the census-takers have listed as veterinarians, but in California, at least, their figures seem fairly accurate. In 1930 we had, according to the census, 563 veterinarians. A corrected list of licensed veterinarians to December 1, 1933, gives us 546. When we take into consideration the fact that most of the federal veterinarians, working in this state, are not licensed, it will bring these figures into close agreement.

As we all know, the list of A. V. M. A. members includes many names of veterinarians who are not residents of the United States. So it would seem that the A. V. M. A. represents only about one-third of the veterinarians in the country. I am not here to criticize what has been done in the past, but merely to point out that something radically different will have to be done if we ever expect to present a united front on any national proposition.

One Forward Step Taken

The greatest fault of veterinary associations is that they have been entirely too loosely knit. We have recently made a step forward in partially uniting the various state associations with our national association. We should go a step farther and unite local associations into one large state and national organization.

National and state associations cannot meet often enough to do effective work. In this day of easy and rapid transportation, local associations could be organized by counties, or groups of counties, somewhat along the lines on which lodges are organized. In this way, a strong national organization could be built up and do effective work for the veterinary profession. Each local association should have a Committee on Public Re-

lations, working with state and national committees of the same character to acquaint the public with the value of veterinary service.

We have been too prone to hide our "light under a bushel." We do not take proper advantage of the publicity channels that are open to us. It is only recently that the public press has learned the proper usage for the term "veterinarian." They have for years insisted on speaking of us as "veterinarys." We are not trained as publicity men. What we need is a national publicity man who understands our problems and who would spend his entire time writing up veterinary activities for the public press. It was left for a dog food manufacturing company in southern California, which featured the veterinarian in page advertisements along with his product, really to bring to the attention of the general public in that section of the state one of the functions of the profession. Newspapers will print news stories dealing with animals if they are properly written. We could have a national press bureau for the collection and dissemination of such material. This could be done without heavy expense to anyone, if we would all get behind such a movement. I believe if we would put such a proposition up to the men of our profession, we could double our membership in a short time.

Our Ranks Not Overcrowded

In many respects, we have been fortunate. The decline in horse practice came before the depression so that our members were able to readjust themselves. Now, with the passing of tractor farming, it seems probable that part of this branch of our work will be restored. We do not have the overcrowded condition in our ranks that exists in most of the other professions, and consequently have not suffered to the extent that many professions have.

We should profit by the mistakes that have been made by those professions that have practiced "mass production" in turning out graduates. It should not be difficult to make a national survey* to determine the number of new men required in our ranks each year. With our present set-up of veterinary colleges, it should be possible to limit or increase the number of graduates to approximate the demand.

*Such a survey was made by the A. V. M. A. Committee on Education. See report made at the Kansas City convention in 1931, and published in the JOURNAL of the A. V. M. A., November, 1931, pp. 669-702. Table XV of the report indicated that it would require 296 graduates per year to maintain the numerical strength of the veterinary profession at the 1930 figure.

There is a danger in too many or too few graduates. If we do not have sufficient qualified men to do the work necessary in preserving the health of our domestic animals, then the animal industry will suffer. This condition of affairs would give an opportunity for lay inspectors to crowd in and attempt to do the work that should be done by veterinarians. Then, of course, our profession would suffer.

On the other hand, the turning out of too many new men, without places for them, demoralizes our entire set-up. Such a procedure is not for the best interests of the individual, the colleges, or the profession. We cannot expect the public to have much respect for a starving profession. In this country, the people look up to those who can create places for themselves, and are able to live well. A man who can take care of his family, educate his children and do his fair share in supporting worth-while community projects, is a "good citizen." That is what we want our members to be—good citizens. We cannot be good citizens if our ranks become overcrowded, and we have to fight for the very existence of life. We wish the public to look up to and respect us. We cannot expect the public to respect a half-dozen run-down-at-the-heel men in a community who are bickering and fighting one another, to the extent they would two or three men who have business enough so they do not have time to knock their competitors.

Every time a veterinarian fails in a community, it is a reflection on all of us. So we should first see that he has the necessary professional qualifications to make a success, and then, I believe, we should attempt to prevent the overcrowding of his field so that he may have a chance for a fair livelihood.

Distribution of Veterinarians

This brings up the problem of distribution of veterinarians. We have the same problem here that the medical profession has, namely, the crowding of a large number of members into the large cities. The Los Angeles area is a good example of this condition, where we probably have more veterinarians to the square mile than anywhere else in the world.

I am indebted to Dr. Haring for some very interesting figures on the number and location of veterinarians in the United States. I first have the census report which, according to the 1930 figures, gives the total number of veterinarians in the United States as 11,858. In 1920 the total was 13,473; so, according to the census, we lost 1,615 members in ten years. However, look what has

happened in California. The 1920 report gave us 439 veterinarians, while in 1930 we had 563, an increase of 124 men during the ten years.

It is interesting to note the states where there was an increase in the number of veterinarians, during the time in which there was a national decrease. They are Florida, with an increase of 25 men; Maryland, with an increase of 13 men; Minnesota, 39; South Carolina, 14; Virginia, 9; and, as I said before, California, 124.

It is also of interest to list the states which have lost veterinarians during the ten-year period. Illinois was the heaviest loser, with 216 men. Then we have Indiana, with 145; Iowa, with 143; Ohio, 139; Pennsylvania, 139; and Michigan, 117. Consider also the plight of the state of Wyoming, which in 1920 had 34 veterinarians and, in 1930, only 17, a loss of 50 per cent.

An exhibition of stability in veterinary population such as we should all strive for is furnished by the state of Nebraska, which in 1920 had 436 veterinarians and, in 1930, had 435, a loss of only one man.

The distribution of veterinarians in the state of California shows that veterinarians, like physicians, are flocking to the large cities. The San Francisco Bay area has 94 licensed men, while Los Angeles has 156.

Areas Without Veterinary Services

We have large areas of the country that are deprived of veterinary service. On the other hand, we have areas that have reached the saturation point as far as veterinarians are concerned. Los Angeles County is one of the best examples of this condition of affairs.

A man, who had recently visited a southern state, made this significant statement. He said: "There is no hog business in that state any more. The hogs have all died of cholera." I asked if they were not vaccinating the hogs in the state and he said: "Oh, the county agents handle all the vaccinating now, but that hasn't saved the hog industry."

It seems to me that such states could well afford to subsidize a veterinarian in such areas to take control of contagious diseases of live stock. Here is where we need an educational campaign. A good many people in the rural sections are looking to the wrong source for their information and advice on the control of disease.

After all is said and done, it is the individual who creates a favorable or unfavorable impression upon the general public. It is the man on the firing line in his daily contacts with the public who has the most influence in building up a favorable regard for the veterinary profession. One of the first things we should do to raise the standard of the veterinary profession is to see that incompetent and irresponsible men are weeded from our ranks.

If we expect to maintain the veterinary profession upon a high plane, we should see that veterinary examining boards are divorced from politics by amending practice acts so that appointments could be made only from a list submitted by some responsible association, or that a candidate, to qualify for appointment, would be required to file a petition of approval signed by at least fifty licensed veterinarians.

It is a sad state of affairs in a country founded upon the principle of democracy, when one man who may know nothing of the problems involved, may make appointments affecting the welfare of a profession. Such appointments made through political expediency tend to destroy the morale of our membership and break down the enforcement of veterinary practice acts.

Internships for Veterinarians

In our attempt to raise the standards of the veterinary profession, we should consider a provision of state practice acts requiring a six-month internship with a practicing veterinarian before new graduates are permitted to take a state veterinary examination.

These boys who have spent their entire time in school have had little time to meet the practical problems of veterinary practice. They should have this opportunity of learning how to apply their knowledge before they are cast upon their own resources. It should prove no hardship to them and would have a tendency to unify the profession.

Another thing that should be done is to make a careful inquiry into the past records of all applicants for a veterinary license before they are permitted to take an examination. This would apply principally to those moving from state to state. We would have saved ourselves a lot of grief in this state if this rule had been rigidly enforced in the past.

I feel quite optimistic in regard to the future of the veterinary profession. I believe we are approaching a period of increased demand for veterinary service. There is no question but that the horse and the mule are more popular and more valuable

than for many a year. With the returning interest in the "sport of kings," as exemplified by the building of new race tracks, the breeding and training of race horses should return and become a profitable industry. With the upturn in economic conditions, the large-animal practitioner may expect better business. The dairy and swine industries have passed through trying times, but all signs point to better days ahead.

Small-animal practice has held up remarkably well during the past two years. This is one of the surprises of the times, as this branch of our profession, catering to a special class, would be expected to suffer the most. Apparently this has not been the case. With a constantly increasing interest in small-animal pets by the public, as indicated by continued heavy sale of prepared dog and cat foods, this branch of the profession can be expected to go forward.

Veterinary practice is serious business. Veterinarians do not belong to the jazz age. With the passing of this wild stage in our national history and the return to sane and sensible living, the veterinarian should come into his own.

Our problems are then, as I see them:

First, to create a strong national veterinary association.

Second, to regulate the supply of veterinarians to approximate the demand.

Third, through giving publicity to actual conditions, to attempt to regulate the distribution of veterinarians to prevent overcrowding.

Fourth, to protect our members from chiseling commercial and other organizations which are constantly trying to infringe upon the field of the veterinarian.

Fifth, to keep our members in line, by preventing unfair and unethical methods of practice.

Sixth, to strive constantly to improve ourselves—individually and collectively, to uphold the honor and respect of the veterinary profession.

Kansas Board Reorganized

The Kansas State Board of Veterinary Examiners met in special session at Topeka, July 6, 1934, for the purpose of reorganization. Those in attendance at the session were: Dr. E. G. L. Harbour, of Lawrence; Dr. J. A. Bogue, of Wichita, and Dr. Thomas P. Crispell, of Parsons. Dr. Bogue was elected President, and Dr. Crispell, Secretary, to serve during the coming year.

A REPORT OF THE GIANT NEMATODE, DICTOPHYME RENALE, FROM A DOG, WITH A SUMMARY OF AMERICAN RECORDS*

By PAUL C. UNDERWOOD *and* WILLARD H. WRIGHT
Zoölogical Division, U. S. Bureau of Animal Industry
Washington, D. C.

During a necropsy performed recently on a male German Shepherd dog 1½ years of age, the writers discovered two live male specimens of *Dioctophyme renale* in the peritoneal cavity. The dog was obtained from Norfolk, Va., for experimental treatment of *Dirofilaria immitis* infestation and had been destroyed for the purpose of observing the effect of the treatment.

The worms were found coiled between the lobes of the liver and under the gall-bladder. They measured 20 and 26 cm, respectively, in length. A viscid, red-brown, sero-fibrinous exudate covered the liver, a small area of the omentum, and the ventral portion of the diaphragm.

The left kidney was hypertrophied to about one-half more than its normal size but otherwise was in good condition except for a slight fibrosis in the cortex. Only vestigial remains of the right kidney could be found. These remains consisted of a mass of fat in which was embedded a small fibrous mass about 4 cm in length. Incision of the fibrous mass revealed it to be the degenerated remains of the right kidney. The hilus of the degenerated kidney was present and in open communication with the related ureter. The lumen of the ureter was partially occluded and thickened from chronic inflammatory changes in the wall. In the fibrous portion of the kidney remains were two pea-sized, caseous nodules which contained greenish-yellow pus. Buried deeply in what had formerly been the cortex was a small lead pellet the size of a No. 6 shot.

From a clinical standpoint this dog appeared to be in good condition and had gained considerable weight following the heart-

*Received for publication, May 7, 1934.

worm treatment. It is worthy of note that the Fouadin treatment for the heart-worm infestation had no apparent effect on *D. renale*.

There have been a number of reports published on the occurrence of *D. renale* in dogs in the United States and Canada, and it appears from these reports that the worm, while comparatively rare, is somewhat generally distributed. Although the literature shows very few cases reported, there have, no doubt, been many cases which have gone unrecorded. That the parasite is not so rare as commonly supposed, is concluded after discussions with veterinary practitioners who have encountered it during necropsies of dogs in an undetermined number of cases over a period of years in their practices. These cases have not been recorded in the literature.

The U. S. National Museum Helminthological Collection contains specimens from nine cases of this parasite in dogs, of which no published report has been made as far as the writers have been able to determine. Four of these cases originated from North Carolina, two from Washington, D. C., one from Maryland and two from South Carolina. In six of these cases the worms were located in the abdominal cavity; in one case the worm was recovered from the pericardium. There is no record of the location of the parasites in two of the cases.

Riley,[1] in 1916, reported 27 definite cases in the United States and Canada. Of these, 19 were published for the first time, while eight were already on record. Twelve of these 27 cases related to the occurrence of the worm in the peritoneal cavity. The same year Hall[2] reported three cases: one from Baltimore, found by Halsted; the second found by Baker, at Chicago, and the third found by La Rue, at Ann Arbor, Michigan. Later, Hall[3] added two cases found by him in Detroit dogs and mentions that Brenton had seen about half a dozen cases of the occurrence of this worm in dogs in Detroit. Following the publication of this record by Hall, he received a communication from Ralph W. Nauss, stating that during Nauss' student days at Northwestern University Medical School, between 1902 and 1905, *D. renale* was found frequently in dogs used at that institution. Nauss mentions another case at the same institution in 1911.

Maxfield[4] reported *D. renale* from a dog, in Iowa, in which the worm was present in the abdominal cavity. Stiles, in 1924, in a personal communication to Hall, mentioned two cases, one from a dog in Baltimore, and the other from a dog in Washington, D. C.

Riley,[5] in 1917, added one case to those previously reported by him and in 1925[6] he listed three additional cases. Wright,[7] in 1930, reported finding two of these parasites in the peritoneal cavity of a dog at Elkton, Maryland. Secord[8] states that two cases have been found in Ohio. Swales[9] lists *D. renale* from dogs in Ontario and Quebec and states that the parasite is common in northern dogs. However, he gives no information as to the number of cases actually brought to his attention.

Hall[3] has summarized 38 cases of *D. renale* infestation in the United States and Canada up to 1917; this summary includes his two Detroit cases and Riley's 1917 case. Adding to this number the nine cases represented in the U. S. National Museum collection, Stiles' two cases, Maxfield's case, Riley's three cases, and those of Wright, Secord and the present case, we have a total of 57 cases accurately recorded. Swales' report is difficult to interpret from the standpoint of the number of cases represented. Likewise, the report of Nauss gives no definite information as to the number of times the worm actually was found. However, it would seem that a total of 75 cases would offer a conservative estimate of those represented in the reports brought together here. Probably this number represents only one-half or less of the dogs in the United States and Canada from which *D. renale* actually has been recovered.

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A sign, seen in a window recently, reads: "Piano lessons. Special pains given to beginners."

CYSTIC KIDNEY IN A COMMON MALTESE CAT*

By J. F. BULLARD, *LaFayette, Indiana*
Purdue University Agricultural Experiment Station

This cat was brought to the laboratory to be destroyed, as the owner said the cat had been examined by a veterinarian and a diagnosis of abdominal tumor made.

The cat had been sick for a week. Up until that time it apparently had been normal. The present examination revealed an extremely emaciated condition and a rough hair-coat. The breath

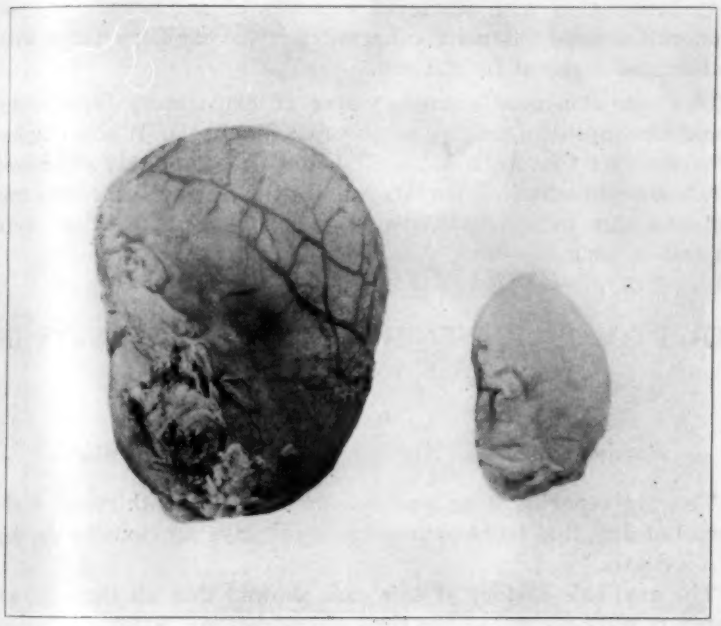

FIG. 1. Comparative size of the left cystic kidney and the right fibrosed kidney.

was very fetid and the cat was in a state or coma. A firm but slightly movable mass, about the size of a hen's egg, could be palpated in the sublumbar region.

The postmortem examination revealed that the kidneys were the seat of the trouble. The left kidney was greatly enlarged. It measured 6.5 by 9 by 5 cm. There was no evidence of any normal kidney tissue present. The entire mass was one large cyst. No

*Received for publication, June 22, 1934.

evidence of an occlusion or stenosis could be found in the remainder of the urinary tract. The right kidney was much smaller. Its dimensions were 3 by 5 by 2 cm. It was grayish white in color and showed an irregular surface with many contractions and scarred areas. The capsule was thickened and very adherent.

No microscopic examination was made of the left kidney. The right one showed an extensive chronic interstitial nephritis with the fibrosis especially abundant in the cortex. This accounted for the roughened, pitted surface of the kidney as seen on gross examination. A very large number of glomeruli were fibrosed and scarred. Many of the uriniferous tubules showed degenerative fatty infiltration with scattered amounts of amylodosis. Other glomeruli showed extensive congestion of the capillary tufts with well-defined crescent formations.

This case is a good example where an exploratory laparotomy would have aided in making an accurate diagnosis. It also emphasizes the fact that both kidneys should be thoroughly examined before a nephrectomy is performed. In this instance the case was hopeless, due to the extensive pathological changes that were present in both kidneys.

STREPTOCOCCIC VEGETATIVE ENDOCARDITIS IN A SMALL PIG*

By J. F. BULLARD, *LaFayette, Indiana*
Purdue University Agricultural Experiment Station

The pig reported here was one of a group of thirteen, eight weeks of age, that had been castrated six days previous to the day of its death.

The available history of this case showed that all the pigs apparently were normal at the time of castration. After castration, all did well with the exception of one which became listless and weak, developed anorexia and remained by itself.

At the time of autopsy, the pig appeared to be in a fairly good state of nutrition. External examination revealed that the castration wounds were not healing.

In the abdominal cavity small blood-clots were seen in the sublumbar region which apparently resulted from the castration. A mild fibrinous peritonitis was seen on the colon and small intestines.

*Received for publication, June 22, 1934.

The main pathological lesions were confined to the heart. It was slightly enlarged and rather flabby. When opened, a very extensive, yellowish-white vegetation was found completely involving the tricuspid valve. Extension of smaller vegetations included the cordae tendinae. A large portion of the apex of the ventricle was occupied by the mass, which was hard and firm. Considerable deposition of lime salts was found in these vegetations.

The adjacent bronchial lymph-glands were cultured and a non-hemolytic, short-chained streptococcus was isolated which was

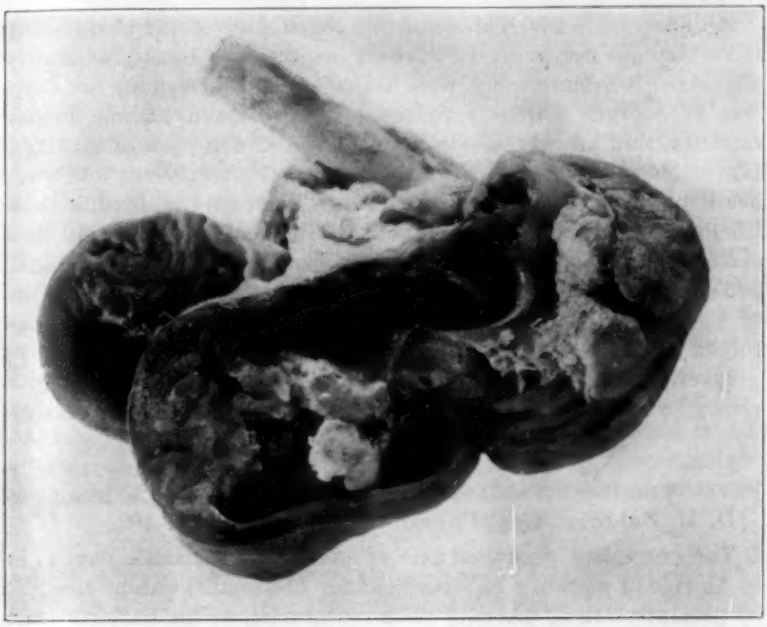

FIG. 1. Extensive vegetations on the tricuspid value of the heart of a pig.

non-pathogenic for guinea pigs in subcutaneous inoculations and for rabbits by the intravenous route.

The accompanying photograph (fig. 1), a right lateral view, shows the exposed right auricle and ventricle with the extensive vegetative growth on the valve.

It is quite evident, due to the presence of lime deposits, that this condition was present before castration was done. This condition no doubt was the cause of the pig not doing well after castration and ultimately resulted in death.

BLOOD CHEMISTRY AND ADRENAL INSUFFICIENCY IN CATS. Raymund L. Zwemer and Ruth C. Sullivan. Endocrin., xviii (1934), 1, p. 97.

An analysis is given showing the chronology of the blood chemistry changes occurring in adrenal insufficiency in approximately 45 cats. Adrenalectomy was found to be followed by an early loss of sodium which is reflected in a lowered carbon dioxide capacity, and an increase in potassium and non-protein nitrogen. Lower blood sugar and chloride and higher phosphorus and magnesium values also appear before death from adrenal insufficiency. Chemically, the syndrome of adrenal insufficiency seems to be a disturbance of salt and water metabolism, with loss of water and depletion of sodium and blood chloride together with an increase of potassium in the blood. Injection of an extract of adrenal cortex into adrenalectomized animals was found to bring about a reversal of the chemical changes occurring in adrenal insufficiency in addition to affecting the observable symptoms favorably.

REVERSIBLE INACTIVATION OF BACTERIOPHAGE. A. P. Krueger and D. M. Baldwin. Gen. Physiol., xvii (1934), 4, p. 499.

The complete inactivation of antistaphylococcal phage by bichloride of mercury (2.8 per cent for 216 hours) can be reversed by precipitation of mercury with restoration of the bacteriophage to its original titre. This behavior seems more compatible with the known properties of certain enzymes than with those of living protoplasm.

THE EPIDEMIOLOGY OF PARASITIC GASTRITIS IN SHEEP. E. L. Taylor. Jour. Agr. Sci., xxiv (1934), 2, p. 192.

There are two epidemiological types of parasitic gastritis: (1) where pathogenic infestation is acquired from a residue of infective larvae present on the land; (2) where pathogenic infestation is acquired as a result of autoinfestation. Infective larvae of Ostertagia can regain the surface of the soil after being turned

in by the plow and retain their infectivity for eight to ten months in sufficient numbers to cause severe disease. Nematodirus larvae treated similarly can regain the surface and retain their infectivity *en masse* throughout the winter months. Severe haemonchosis can develop by autoinfestation alone, on clean, arable land in as short a time as seven weeks. Severe ostertagiasis can develop from residual soil infestation alone, in as short a time as four weeks.

The residue of infective larvae where sheep have been penned over a field may be different in different parts of the field. Lambs up to seven weeks old are not susceptible to haemonchosis under natural farming conditions. Conditions may be so favorable for larval development on arable land that even the relatively small number of eggs disseminated by the ewes may be sufficient to have a dangerous infection in the soil after once penning over. Six days is sufficient for the mass development of infective larvae under actual field conditions. Bare pastures are a danger as conditions are then optimum for the acquisition of the larvae. Grazing where there is an abundance of forage to such an extent that the lambs cannot eat more than half of the crop does not safeguard against development of ostertagiasis on arable land. There is an enormous difference between the suitabilities of various field conditions for larval development. An extra supply of concentrated food is sufficient to ward off an outbreak of the disease where the rate of infestation only reaches a certain level, but where infestation is high, a plentiful supply of concentrated food does not prevent the development of a severe outbreak.

THE INFECTION OF FERRETS WITH SWINE INFLUENZA VIRUS. Richard E. Shope. Jour. Exp. Med., lx (1934), 1, p. 49.

Earlier observations of Smith, Andrews and Laidlaw that swine influenza virus is pathogenic for ferrets when administered intranasally is confirmed. A disease that is clinically more severe and pathologically more extensive is obtained if inoculation with the virus is performed under ether anesthesia. Animals infected in this way show at autopsy an edematous type of pneumonia of lobar distribution which may terminate fatally. The virus maintains its pathogenicity for ferrets when stored in 50 per cent glycerol at refrigerator temperature for as long as 75 days. After serial passage through 16 ferrets, the virus is still capable of inducing swine influenza when mixed with *H. influenza suis* and administered intranasally in swine. Ferret passage causes no

apparent attenuation of the virus for swine. Serum from pigs recovered from swine influenza is capable of neutralizing the ferret-passaged virus for either swine or ferrets. Serum from recovered ferrets neutralizes the swine influenza virus for either ferrets or swine.

UNDULANT FEVER IN NEW YORK STATE. Ruth Gilbert and Marion B. Coleman. Jour. Inf. Dis., liv (1934), 3, p. 305.

If organisms of the abortus-melitensis group can be divided into subspecies or types, the authors found that only one—the bovine type—is prevalent in the relatively large area served. The information suggests that cattle or dairy products have been the source of the infection in most of the cases of undulant fever studied. A large percentage of the patients have had no contact with cattle, but have used raw milk or cream. It is suggested that general and efficient pasteurization might effect a marked decrease in the incidence of undulant fever.

CLASSIFICATION OF BRUCELLA. Axel Thomsen. Jour. Inf. Dis., liv (1934), 3, p. 345.

Danish *Brucella suis* strains grown in 200-cc bottles of beef broth in the incubator, completely undisturbed for a period of 1 to 3 weeks, show the development of an abundant surface growth, difficult to homogenize by shaking and which broke and quickly settled to the bottom after slight jarring. Two American porcine strains acted in a similar manner. Twenty-five bovine strains grown in the same manner produced only a slight surface growth. Five bovine strains which would grow only under carbon dioxide produced an abundant surface growth. Six melitensis strains yielded a vigorous diffuse growth with a moderate sediment, presenting degrees between the porcine and the bovine types.

TESTS FOR CROSS-IMMUNITY BETWEEN THE VIRUS OF BORNA DISEASE AND THAT OF EQUINE ENCEPHALOMYELITIS. Beatrice F. Howitt and K. F. Meyer, Jour. Inf. Dis., liv (1934), 3, p. 364.

There is apparently no immunity to Borna disease in susceptible animals that have recovered from the California encephalomyelitis or have shown immune reactions to the latter, while the giving of injections of Borna disease virus to guinea pigs does not render them immune to the other strain. The live viruses seem to be immunologically distinct. Rabbits immune to Borna disease and tested with the virus of encephalomyelitis seemed to

have attained a degree of immunity. It is concluded from certain clinical, histologic and immunologic observations that it is logical to assume that the equine encephalomyelitis described in California is distinct from the condition known by European writers as Borna disease.

IMMUNIZATION OF GUINEA PIGS TO THE VIRUS OF EQUINE ENCEPHALOMYELITIS. Beatrice F. Howitt. Jour. Inf. Dis., liv (1934), 3, p. 368.

Suspensions of virus attenuated by chloroform, phenol, glycerite of phenol, or solution of formaldehyde, respectively, were largely unsatisfactory for the active immunization to the virus of equine encephalomyelitis although immunity was always present in the animals that survived the intracerebral injections of live virus. Successful immunization without fatalities due to vaccination was obtained in several groups of guinea pigs by the simultaneous administration of serum and virus followed by one or two doses of suspension of active virus alone. Immunization may be obtained by small doses of live virus alone when given intramuscularly, subcutaneously or intradermally, although fatalities due to vaccination may occur. Immunity may be retained in guinea pigs for at least six or seven months after the animals receive the last injection of virus, even though only one dose is given.

THE FLOW AND COMPOSITION OF LYMPH IN RELATION TO THE FORMATION OF EDEMA. A. A. Weech, E. Goettsch and E. B. Reeves. Jour. Exp. Med., lx (1934), 1, p. 63.

The capacity of the lymphatics for removing fluid from the tissues greatly exceeds the rate at which freshly formed tissue fluid can be made available for removal. Edematous regions can be rendered non-edematous by the application of measures such as massage, passive motion, or normal exercise, which activate the lymphatics. During continuous activity the rate of lymph-flow is first variable and later relatively constant. Lymph formation in the edematous animal is only slightly greater than under conditions of normality. The ratio of serum protein to lymph protein is greater in the edematous than in the normal animal. Differences in tissue pressure exist between the states resulting from quiescence and activation of the lymphatics. When the lymphatics are activated, it is possible that capillary reabsorption may be completely in abeyance. A decline in the proteins of the plasma may be associated with a diminished permeability of the capillaries.

EXPERIMENTAL STUDIES OF POSTMORTEM BACTERIAL INVASION IN ANIMALS. Caspar G. Burns. Jour. Inf. Dis., liv (1934), 3, p. 388.

B. welchii, *B. coli* and staphylococci are capable of invading the tissues of animals within from four to 48 hours after death when the bodies are kept at 25° C. A large group of pathogenic and non-pathogenic bacteria failed to invade the tissues after death, even though ample opportunity was given for invasion to occur. The quantity of organisms, their location within the body and the time between death and autopsy are undoubtedly important factors influencing postmortem invasion. A temperature of 10° C. inhibits the growth of *B. coli* and *B. welchii*, even after incubation for 96 hours. Increased temperature failed to bring about spread of strains of bacteria known to be non-invasive.

STUDIES OF THE ETIOLOGY OF SPONTANEOUS CONJUNCTIVAL FOLLICULOSIS OF RABBITS. I. Transmission and filtration experiments. Peter K. Olitsky, Jerome T. Syverton and Joseph R. Tyler. Jour. Exp. Med., lx (1934), 1, p. 107.

Spontaneous conjunctival folliculosis of rabbits exists in two forms: type I in which the lesions are localized and the disease is relatively inactive, and type II, in which the follicles are closely distributed over the entire surface of the conjunctivae and the affection is more active and characterized by extensive inflammatory reactions. One type can be converted into the other, either by experimental methods or by natural processes. The disease can be transmitted from rabbit to rabbit by means of subconjunctival inoculation of suspensions of the affected tissues or by instillation of such material into the conjunctival sac or by mere contact of folliculosis animals with rabbits having smooth conjunctivae. The causal agent is not filtrable through Seitz discs or through Berkefeld V candles. The lesions are characterized by a persistent and progressive chronicity and show certain resemblances to the granulomata.

SECRETIN. Its influence on the amount of hemoglobin in the circulating blood. John Ferguson. Endocrin., xviii (1934), 2, p. 188.

Twenty-four rabbits were injected subcutaneously with a secretin preparation and six rabbits with a secretin concentrate preparation. The hemoglobin of a unit volume of the circulating blood was determined before the injection, and at intervals of

20, 45, and 80 minutes after injection. The mean maximum percentage increase in the first group was 5.8 and in the second group 5.4. The secretin preparation appeared to act more slowly than the secretin concentrate but its effects lasted longer. More consistent results were obtained with the secretin concentrate than with the secretin preparation.

AN ATTEMPT TO DUPLICATE CERTAIN HORMONE RESPONSES BY MEANS OF DRUGS. M. C. D'Amour. Endocrin., xviii (1934), 2, p. 235.

The action of drugs which induce a hyperemia of the genital organs was studied in an attempt to determine whether the response to the gonad-stimulating hormone of pregnancy urine and to estrin might be due to the hyperemia which they produce. Yohimbine, cantharidin and histamine were injected into groups of six male and six female 21-day-old rats daily for ten days. Noticeable hyperemia was produced, but no increase in the size of the uteri, opening of the vagina, or hemorrhagic follicles. There was no increase in weights of seminal vesicles and ovaries over those of uninjected controls. Yohimbine and cantharidin, given respectively to three and two young female rabbits for four and two days, produced marked hyperemia but no ripe follicles. No estrus smears were obtained when groups of ten ovariectomized rats were injected with cantharidin and yohimbine each day for seven days. The degree of hyperemia which is induced by dosage which can be tolerated is not comparable to that which follows hormone injection.

NERVOUS AND HORMONAL FACTORS IN LACTATION. H. Selye, J. B. Collip and D. L. Thomson. Endocrin., xviii (1934), 2, p. 237.

Established lactation was rapidly and invariably terminated by hypophysectomy in six mice, in a large series of rats, and also in 22 mice and 12 rats which were simultaneously pregnant and lactating. Hypophysectomy was carried out on 24 rats, six mice, and one guinea pig during gestation without leading to abortion. All secreted milk for a few hours after parturition. Cesarean section late in pregnancy induced lactation in 16 rats but not in six others, in which hypophysectomy was also carried out, or in eight others, in which the uterus was distended with paraffin after removal of the foetuses. The correlation between the pregnant uterus, the ovaries, the hypophysis and the mammary glands is discussed in the light of these and other findings.

Regular Army

Major Forest L. Holycross is relieved from assignment and duty at the Ohio State University, Columbus, Ohio, and directed to report to the commanding officer Fort Hayes, Ohio, for duty. He will also report to the commanding general Fifth Corps Area and the commanding officer general reserve depot Columbus, Ohio, for duty in addition to his other duties.

The promotion of Captain Frank M. Lee to the grade of major to rank from May 16, 1934, is announced.

Major Jack G. Fuller is relieved from assignment and duty at Cornell University, Ithaca, N. Y., to take effect on or about August 1, 1934, will then proceed to Fort Snelling, Minn., and report to the commanding general for duty.

Captain Herbert M. Cox is relieved from duty at Plattsburg Barracks, N. Y., effective in time for him to proceed to New York, N. Y., and sail on the transport scheduled to leave that port on or about October 4, 1934, for the Panama Canal Department.

First Lieut. Ralph W. Mohri is relieved from further detail as a student, The Cavalry School, Fort Riley, Kan., effective upon completion of his present course of instruction, and will report to the commanding general, Fort Riley, Kan., for duty.

Lt. Colonel Christian W. Greenlee is relieved from assignment and duty at Fort Benjamin Harrison, Ind., at such time as will enable him to proceed to New York, N. Y., and sail on the transport scheduled to leave that port on or about October 4, 1934, for the Panama Canal Department. Upon arrival at Panama will report to the commanding general for assignment to duty with the Veterinary Corps.

Major Raymond I. Lovell, is relieved from duty at Fort Snelling, Minn., at such time as will enable him to proceed to New York, N. Y., and sail on the transport scheduled to leave that port on or about October 4, 1934, for the Panama Canal Department. Upon arrival at Panama he will report to the commanding general for assignment to duty with the Veterinary Corps.

Major James L. Barringer is assigned to duty at Plattsburg Barracks, N. Y., effective upon completion of his present tour of foreign service in Panama.

Veterinary Reserve Corps

Promotions

To

Arundel, Hugh Francis Jones 1st. Lt..Savannah Ave., Statesboro, Ga.
German, Walter Allen.....1st. Lt..5346 Cornell Ave., Chicago, Ill.
Lewis, Henry Vance.....1st. Lt..2027 Davie St., Davenport, Iowa.

TWELFTH INTERNATIONAL VETERINARY CONGRESS

Waldorf-Astoria Hotel, New York, N. Y. August 13-18, 1934

OFFICERS

Chairman of the Organizing Committee: Dr. A. Eichhorn.
Vice-Chairman: Dr. L. A. Merillat.
Treasurer: Dr. John R. Mohler.
General Secretary (to whom all communications should be addressed):
Dr. H. Preston Hoskins, 221 N. La Salle St., Chicago, Ill.

Membership Campaign

New York still leads in the membership campaign and is well on toward the 300 mark. California has a firm hold on second place, while Pennsylvania is the third state to pass the 100 mark and seems comfortably settled in third place. Ohio continues in fourth position, and is near the century mark. Illinois, in fifth place, is within striking distance of a membership list of 100. Minnesota passed Missouri and now occupies sixth position. New Jersey jumped from tenth to seventh in the list, while Iowa advanced from 15th place to ninth. Nebraska appears in 14th place, an achievement that represents fine activity in that state, since this is the first time Nebraska has appeared in a list of the leaders. Texas is now 19th in the list. The figures compiled up to and including July 24 show the following 32 leaders in the membership race:

New York	269	Connecticut	30
California	159	Indiana	29
Pennsylvania	119	Texas	29
Ohio	95	Maryland	22
Illinois	89	New Hampshire	22
Minnesota	76	Washington	22
New Jersey	73	Florida	21
Missouri	69	Michigan	21
Iowa	69	Vermont	19
Massachusetts	59	Virginia	17
District of Columbia	56	Oregon	15
Wisconsin	53	Nevada	12
Colorado	46	West Virginia	11
Nebraska	43	Arizona	10
Oklahoma	40	Louisiana	10
Kansas	33	North Carolina	10

Twenty-two foreign countries are represented by five or more members. Belgium appears on the list for the first time, and

takes third place, surpassed only by Germany and Great Britain and Northern Ireland. Yugoslavia enters the list with 38 applications received, and takes fourth place. Other countries that appear on the list for the first time are: Cuba, with seven members; Poland, with seven; Peru, with six; and Austria and Norway, with five each. Twenty-four other countries, with one, two or three members each, have signified their interest in the Congress, in addition to the 22 listed here:

Germany	125	Egypt	13
Great Britain and Northern Ireland	73	Japan	13
Belgium	44	Switzerland	11
Yugoslavia	38	Australia	10
Canada	28	Union of South Africa	9
Chile	27	Sweden	8
France	25	Cuba	7
Netherlands	18	Poland	7
Spain	17	Peru	6
Dutch East Indies	13	Austria	5
		Irish Free State	5
		Norway	5

Finances

Nine additional contributions have been received from veterinary organizations during the past month. Of these, the largest is that of $500 given by the New York State Veterinary Medical Society. A Canadian group appears in the list for the third time, the two other Canadian contributors being the Ontario Veterinary Association and the Veterinary Association of Saskatchewan. The Veterinary Medical Association of New York City appears for the second time, the first contribution of $100 having been noted in the February issue of the JOURNAL.

The list is as follows:

East Tennessee Veterinary Medical Association	$ 10.00
New York City, Veterinary Medical Association of	140.10
New York State Veterinary Medical Society	500.00
Northeastern Pennsylvania Veterinary Medical Club	50.00
Oregon Veterinary Medical Association	50.00
Quebec, College of Veterinary Surgeons of the Province of	50.00
Schuylkill Valley Veterinary Medical Association	10.00
Texas, State Veterinary Medical Association of	100.00
West Virginia Veterinary Medical Association	25.00
Previously acknowledged	6,336.00
	$7,271.10

Honor Roll

Four new names have been added to the list of veterinarians who have contributed the sum of $100 to the Congress treasury. They are: Dr. Wm. Henry Kelly, of Albany, N. Y.; Dr. C. G. Rohrer, of New York, N. Y.; Dr. T. A. Sigler, of Greencastle,

Ind., and Dr. Cassius Way, of New York, N. Y. With these four new additions, the honor roll now boasts of 28 names.

Cuba to Send Dr. Crespo

Dr. Bernardo J. Crespo y García, of Havana, Cuba, member of the Permanent Commission, will be among the eminent Spanish-speaking veterinarians in attendance at the Congress. Especially noteworthy is the contribution Dr. Crespo has made toward the development and progress of the veterinary profession in Cuba.

DR. BERNARDO J. CRESPO

Dr. Crespo was graduated from the Veterinary School, of the University of Havana, in 1910. Shortly thereafter, he was employed by the Department of Agriculture and, after a brief service, became Chief of the Bureau of Animal Industry of the Department, a position he held until recently. During the last few years, he has been assistant professor at the Veterinary School, of the University of Havana; a member of the National Milk Commission of the Department of Health; a member of the Court of the Civil Service Commission for the examination of veterinarians; vice-president of the past three National Medical Congresses, and director of various national live stock exhibitions. He is honorary president of the National Veterinary Association, of the Association of Breeders of Milk Animals and of the Association of Hog Breeders. Dr. Crespo was the official delegate of Cuba to the Eleventh International Veterinary Congress, held in London, in 1930. At present, he is connected with the firm of Lykes Brothers Co., as director of the Division of

Biological Products. He is also director and proprietor of the journal, *Agricultura y Zootecnia.*

Congress Notes

Nevada has set an enviable record in the membership race. Out of 15 veterinarians in the State eligible for membership in the Twelfth International Veterinary Congress, 12 have filed their applications. This gives Nevada an 80 per cent rating. Can any other state show a comparable record?

Dr. Alex R. Anderson, Jr., of Van Nuys, Calif., probably will be the youngest veterinarian to attend the Congress from among those who were graduated from veterinary colleges in 1918. Dr. Anderson received his degree from Grand Rapids Veterinary College, in 1918, when he was 19 years old.

Prof. Gerhard Forssell has been named to represent another Swedish institution, the Veterinary College of Stockholm. Dr. Forssell is a reporter on "Recent Progress in Veterinary Surgery," and a representative of the Swedish government and of the Association of Swedish Veterinary Surgeons, as reported previously in the JOURNAL.

Dr. H. H. Baur, of Monroe, La., will attend as the official delegate of the Louisiana Veterinary Medical Association.

Dr. J. F. Witter, of Orono, Me., will represent the Maine Veterinary Medical Association.

Capt. J. S. Penrose has been selected as the delegate of the Veterinary Association of Victoria, Australia.

Mr. D. F. Stewart has been appointed assistant delegate from the Queensland Veterinary Association, Queensland, Australia.

Capt. R. Daubney has been appointed official delegate of the government of Kenya, East Africa.

Lt. Col. T. Dunlop Young, of London, England, will represent the government of New Zealand.

Dr. Lionel Bull has been named official representative of the Commonwealth of Australia.

Mr. Mils P. Thorshaug and Prof. Halfdan Holth are the official delegates from Norway.

Hector G. Padilla Dubost and Alfonso Bonilla Lopez have been selected to represent the Mexican Veterinary Military Service.

Dr. Rafael A. Osorio will attend the Congress as the delegate from the Mexican Department of Health.

Dr. Friedrich Weber will represent the German National Health Department.

MARYLAND STATE VETERINARY MEDICAL ASSOCIATION

The summer meeting of the Maryland State Veterinary Medical Association was held at the University of Maryland, College Park, June 28-29, 1934, with about 65 in attendance.

The meeting was called to order by the President, Dr. R. C. Reed, of College Park, who made a short address. Dr. Reed stressed the need of coöperation among the membership and particularly the interdependence of practitioners and those engaged in official capacities. Dr. J. W. Hughes, of Elkton, gave an enlightening discussion of "Legal Aspects of Veterinary Practice," in which he pointed out that there are, apparently, certain responsibilities often neglected that may cause unfortunate legal tangles. A general round-table discussion on equine encephalomyelitis was led by Drs. H. W. Schoening, of the U. S. B. A. I., Washington, D. C., and A. L. Brueckner, of College Park. Recent experimental findings were presented and several field cases were discussed.

Dr. P. C. Underwood, of the Zoölogical Division, U. S. B. A. I., spoke on "The Use of Anthelmintics in Veterinary Practice." He discussed the drugs in common use, their methods of application, and the results to be expected. He emphasized the losses caused by parasites and the need for more diligence in their control. The last paper of the morning, "*Brucella Abortus* Infection of Horses," was presented by Dr. L. J. Poelma, of College Park. The cases reported exhibited a marked lameness of a shifting character. All of these cases were from the practice of Dr. R. V. Smith, of Frederick, who discussed the findings and results from the field point of view.

In the afternoon, several surgical operations on horses were demonstrated by Dr. J. B. Koerner, Jr., of Sykesville, and Lt. Col. J. E. Behney, V. C., U. S. A., Fort Myer, Va., assisted by several of the members. Increasing interest in this type of work was evidenced, and several points of technic were brought out.

In the evening, a get-together banquet was held at the Indian

Springs Golf Club. As a deviation from the usual custom, there were no addresses, speeches, talks or elocution of any kind. The dance floor, card tables and comfortable chairs seemed to be all that was necessary to insure a pleasant evening.

Dr. J. J. Jones, of La Plata, opened the program on the second day with a paper on "Ethics in Veterinary Practice," in which he discussed points concerning ethical advertising and ethical practice-building. Mr. Ben F. Brandon, superintendent of the B. A. I. Farm, at Beltsville, discussed the plans under way for developing the Farm and the Experiment Station. Virtually all of the agencies concerned with live stock, soils, crops and forests are moving important units of their field stations and research offices to Beltsville. Approximately $2,000,000 is being spent in this development. When completed, it will comprise the largest agricultural experiment station in the world. Dr. C. M. Grubb, of Rockville, discussed "Small-Animal Work in General Practice," pointing out that the same care and skill necessary to build a large-animal practice are equally necessary in small-animal work.

Dr. DeVoe Meade, head of the Animal and Dairy Husbandry Departments, University of Maryland, presented a paper on "The Newer Knowledge of Nutrition." He discussed the vitamin and mineral requirements of live stock, and gave out charts to the members, showing the foods that contained the necessary elements. Dr. H. M. DeVolt, of College Park, spoke on "Recent Developments in Poultry Practice." Practitioners, he said, should take a more active interest in the control of poultry diseases, especially in Maryland, where the poultry industry is economically more important than several other fields of live stock work.

Dr. Underwood took charge of the small-animal clinic in the afternoon, pinch-hitting for Dr. E. B. Dibbell, of Baltimore, who could not be present because of an illness in his family. Among other things, Dr. Underwood performed a cecectomy on a dog, discussing and illustrating the technic of the operation. An unusually fine poultry demonstration was presented by Dr. DeVolt, to show many of the poultry diseases encountered in Maryland flocks. Dr. Wm. R. Teeter demonstrated the use of colloidal iodin in the treatment of poultry parasites.

Luncheon was served at the Live Stock Sanitary Service Laboratory, on both days, and offered a splendid opportunity for the members to discuss their mutual problems.

At a short business session, a resolution was approved for presentation to the Surgeon General, expressing the appreciation

of the Association for the large number of Army veterinarians who were present to participate in the program. Dr. J. P. Turner, of Washington, D. C., was elected delegate, and Dr. E. B. Simonds, of Baltimore, was named alternate, to the A. V. M. A. House of Representatives for the ensuing year.

MARK WELSH, *Secretary.*

CENTRAL NEW YORK VETERINARY MEDICAL ASSOCIATION

The annual meeting of the Central New York Veterinary Medical Association was held at Marcellus, June 14, 1934. The meeting was called to order at 2:30 p. m., by the President, Dr. M. W. Sullivan. The Secretary, Dr. W. B. Switzer, read a communication inviting the members of the Association to attend the Twelfth International Veterinary Congress and asking that an official delegate be chosen for the Congress. President Sullivan was unanimously elected to be the delegate.

A splendid program was presented. Dr. M. W. Sullivan gave an interesting case report on his treatment of purpura hemorrhagica in a horse. Two blood-transfusions were given, with the horse making a good recovery. The New York State Veterinary College at Cornell University was well represented on the program. Dr. J. N. Frost read a paper on lameness in the horse, which was generally discussed. Dr. H. J. Milks presented an excellent paper on eczema in the dog. Dr. H. C. Stephenson spoke on vomiting in the dog, his remarks provoking an interesting discussion. Dr. D. W. Baker read a paper on Bang's disease and gave a demonstration of the rapid test. This was followed by a long and enthusiastic discussion.

The annual banquet was held at the Tuscarora Country Club. The Rev. Father Joyce, of Marcellus, was introduced, and his witty address was pronounced altogether delightful. Dr. E. T. Faulder, director of the Bureau of Animal Industry, New York State Department of Agriculture and Markets, was the speaker of the evening, with the subject, "Tuberculin Testing and Physical Examinations." A lively discussion followed.

Officers chosen for the coming year are: President, Dr. E. S. Markham, Constableville; vice-president, Dr. Joseph Mosher, Genoa; secretary-treasurer, Dr. W. B. Switzer, Oswego (re-elected).

W. B. SWITZER, *Secretary.*

NEBRASKA STATE VETERINARY MEDICAL ASSOCIATION

The first annual summer clinic of the Nebraska State Veterinary Medical Association was held at David City, June 28, 1934. More than 150 veterinarians registered, including visitors from Iowa, Kansas and Missouri. The clinic began at 8 o'clock in the morning, and it was that hour in the evening before all cases had been disposed of. The various operations and demonstrations showed clearly that Nebraska veterinarians are keeping abreast of modern progress in veterinary science.

Local arrangements were under the direction of Dr. S. W. Phillips, of David City, president of the Association, whose hospital was the center of the clinic activities. He was assisted by Drs. J. D. Sprague and H. Gross, of David City. The Clinic Committee was composed of Dr. Darrell E. Trump, of Utica, Chairman, who was also in charge of the Swine and Small-Animal Section; Dr. O. H. Person, of Wahoo, who was in charge of the Bovine Section, and Dr. G. H. Leenerts, of Humphrey, who was Chairman of the Equine Section.

Contributing to the success of the program was the x-ray equipment supplied by the General Electric Company for demonstration purposes. Swift and Company and Armour and Company, coöperating with the Omaha staff of the U. S. B. A. I., provided a pathological display which was transported to David City by the Corn States Serum Company in a refrigerated truck. In addition to the hospital facilities, a large tent with amphitheater seats was used to accommodate those in attendance. Noonday lunch was served at a local church, while the ladies enjoyed a picnic lunch and entertainment provided for them at the City Park.

Operations were performed in the Equine Section as follows: Operations for roaring, recumbent and standing; cryptorchidectomy; tenotomy; neurectomy and perineorrhaphy. Demonstrations included: Restraint; spavin and ring-bone treatment by the injection method; spinal anesthesia; stomach-tube demonstration; a demonstration of a new type of balling-gun. In the Bovine Section, operations were performed as follows: ablation of the eye; teat strictures and tumors; cryptorchidectomy; rumenotomy; oöphorectomy in a heifer; amputation of a digit. Demonstrations included the following: autopsy of a case of urinary calculus; diagnosis of acetonemia, and a positive ketone test. The Swine and Small-Animal Section program included the

following operations: cryptorchidectomy; ventral hernia; oöphorectomy in a bitch; oöphorectomy in a female cat; castration of a male cat; removal of the membrana nictitans, and an ectropion operation. Demonstrations consisted of intravenous anesthesia and the application of splints in fractures.

E. C. JONES, *Secretary.*

NORTH CAROLINA STATE VETERINARY MEDICAL ASSOCIATION

The thirty-third annual meeting of the North Carolina State Veterinary Medical Association was a joint affair with the twenty-fifth annual meeting of the South Carolina Association of Veterinarians, at Spartanburg, S. C., July 10-11, 1934.

Dr. Raymond Kitchen, of Sumter, S. C., president of the South Carolina Association, presided at the opening session, while Dr. A. H. Williamson, of Charlotte, N. C., president of the North Carolina Association, opened the afternoon session. Dr. G. A. Roberts, of Christiansted, Saint Croix, Virgin Islands, a former member of the North Carolina Association, gave an address on "Strongylosis," as that disease occurs in the Virgin Islands. "Canine Filariasis" was discussed by Dr. I. M. Hays, of Albany, Ga., and "A General Discussion of the Diagnosis and Control of Poultry Diseases" was presented by Dr. F. D. Patterson, of Auburn, Ala.

A banquet brought the first-day program to a close. Dr. Wm. Moore, State Veterinarian, Raleigh, N. C., presided as toastmaster. The speaker of the evening was Dr. C. C. Norton, of Wofford College, Spartanburg, who made an interesting address. A dance followed the banquet.

On the morning of the second day, Dr. C. E. Salsbery, of Kansas City, Mo., read an excellent paper on "Mastitis." A paper, "Judging Dogs and Dog Shows," prepared by Dr. W. T. Scarborough, of Raleigh, was read by Dr. A. A. Husman, B. A. I. Inspector-in-Charge, Raleigh, in the absence of Dr. Scarborough. Dr. Patterson closed the morning session with a talk on "Fundamental Principles in the Diagnosis of Canine Diseases." The afternoon was devoted to a clinic, held at the hospital of Dr. M. R. Blackstock.

The North Carolina State Veterinary Medical Association elected officers for the coming year, as follows: President, Dr. P. M. Abernethy, Burlington; first vice-president, Dr. W. A. Carter, Weldon; second vice-president, Dr. S. B. Till, Biltmore;

secretary-treasurer, Dr. J. Howard Brown, Tarboro (reëlected); directors, Drs. R. E. Taylor, Hendersonville, and H. C. Rea, Charlotte. Dr. A. A. Husman, of Raleigh, was elected an alternate delegate to the A. V. M. A. House of Representatives. Dr. L. J. Faulhaber, of Raleigh, was elected delegate one year ago.

The South Carolina Association of Veterinarians did not elect officers at this time.

The North Carolina Association will meet with the Virginia State Veterinary Medical Association, at Richmond, Va., in 1935.

J. HOWARD BROWN, *Secretary.*

VIRGINIA STATE VETERINARY MEDICAL ASSOCIATION

The forty-first annual meeting of the Virginia State Veterinary Medical Association was held at the Princess Anne Hotel, Virginia Beach, July 12-13, 1934. Forty-four members and their families were present.

The meeting was called to order by the President, Dr. E. H. Drake, of Leesburg, who made the opening address. The literary program consisted of the following papers: "History and Alumni of the United States Veterinary College," by Dr. C. J. Robinson, of Washington, D. C.; "Recent Legislation in Virginia Affecting the Veterinary Profession," by Dr. H. L. Lyon, of Hillsville; "Encephalomyelitis," by Dr. M. S. Shahan, of Washington, D. C.; "Calcium in Clinical Medicine," by Dr. L. E. Starr, of Blacksburg; "A Practitioner *versus* Bang's Disease," by Dr. W. H. Grubb, of Purcellville; "Name It and You Can Have It," by Dr. W. L. Bendix, of Richmond; "Preventive Medicine: Its History, Scope and Future," by Dr. Taylor P. Rowe, of Richmond. A banquet was held in the evening.

An address on "Plans for the Future," by Dr. H. C. Givens, State Veterinarian, Richmond, opened the second-day program. A business session followed. Dr. I. D. Wilson, of Blacksburg, was elected delegate to the A. V. M. A. House of Representatives and Dr. P. M. Graves, of Culpeper, was elected an alternate, both for a period of two years. Officers named to serve during the coming year are: President, Dr. Taylor P. Rowe, Richmond; first vice-president, Dr. I. P. Gilbert, Courtland; second vice-president, Dr. C. J. Robinson, Washington, D. C.; secretary, Dr. I. D. Wilson (reëlected); treasurer, Dr. R. E. Brookbank, Richmond, (reëlected).

I. D. WILSON, *Secretary.*

NECROLOGY

JOHN J. CULP

Dr. John J. Culp, of Ionia, Mich., was found dead in a pasture near his home, June 26, 1934. Death was due to a heart attack, which came as Dr. Culp was pasturing his Shetland ponies for the night. He was nearing his 80th year.

A graduate of Ontario Veterinary College, class of 1887, Dr. Culp was one of the oldest veterinarians in Michigan. His hobby was trained ponies, which were the delight of the youngsters of Ionia. Dr. Culp had gathered his ponies from circuses and elsewhere, and had trained many of them himself. He staged impromptu performances for the children, almost daily. Although Dr. Culp was widely known throughout the State, most of his practice was conducted in and around Ionia. He is survived by his widow.

GLENN A. METCALF

Dr. Glenn A. Metcalf, of Knoxville, Tenn., died at Saint Mary's Hospital, June 29, 1934, following an operation which had been performed a week previous.

Born in Monroe, Kan., July 29, 1878, Dr. Metcalf moved to Knoxville with his parents when he was ten years old. He was graduated from the United States College of Veterinary Surgeons in 1908 and, with the exception of six years spent in government service in Washington, D. C., had practiced his profession in Knoxville since that time. For many years, Dr. Metcalf had been associated with Dr. M. Jacob in conducting the Knoxville Veterinary Hospital.

Dr. Metcalf joined the A. V. M. A. in 1916. He was a member of the Tennessee Veterinary Medical Association, of the East Tennessee Veterinary Medical Society, President of the State Board of Veterinary Medical Examiners of Tennessee and a leader in the veterinary profession of the State. He was a member of the Elks and Odd Fellows lodges. Dr. Metcalf was active

in civic affairs and was prominently identified with many movements for the betterment of the community. Surviving are his widow (née Elizabeth Allen), one daughter, his mother, two sisters and one brother. M. J.

FRANK D. LANDON

Dr. F. D. Landon, of Great Barrington, Mass., died at his home, July 15, 1934, from heart disease.

Born in Delhi, N. Y., September 20, 1877, Dr. Young was a graduate of the U. S. College of Veterinary Surgeons, class of 1903. Following his graduation, he established himself in Great Barrington, and built up an extensive practice through the Southern Berkshire territory. For a number of years, he had done considerable work in connection with the state and federal efforts to control tuberculosis in cattle. He also had engaged extensively in farming.

Dr. Landon joined the A. V. M. A. in 1915. He was also a member of the Massachusetts Veterinary Association. He held membership in the Sheffield Grange, Cincinnatus Lodge of Masons, Order of the Eastern Star and the Elks. Surviving are his widow (née Alice M. Davis), two sons, three daughters and one sister.

EMORY H. THORNBURGH

Dr. Emory H. Thornburgh, of Lynn, Ind., died at his home, July 17, 1934, at the age of 61. He was a graduate of the Indiana Veterinary College, class of 1912. Surviving Dr. Thornburgh are his widow, three sons, three sisters, and three brothers.

PERSONALS

MARRIAGES

Dr. Fritz Volkmar (O. S. U. '31), of Columbus, Ohio, to Miss Irene Louise Frederickson, of Fargo, N. Dak., in Chicago, Ill., June 15, 1934.

Dr. Walter J. Kiff (McK. '16), of Michigan City, Ind., to Miss Loretta A. Sreman, of Burlington, Wis., in Michigan City, June 30, 1934.

Dr. F. G. Caslick (Corn. '30), of Morristown, N. J., to Miss Lillian W. Little, May 8, 1934.

Dr. J. P. Oldsen (Iowa '30), of Rockford, Iowa, to Miss Ruth Dunkelberg, of Rockford, in Rockford, June 30, 1934.

DR. THOMAS MOORE (Ont. '29), of Ottawa, Ont., to Miss Edna Pears, of Guelph, Ont., in Guelph, June 16, 1934.

DR. JOHN M. BAKER (Ont. '32), of Saint Walburg, Sask., to Miss Irene Babineau, of Paradise Hill, Sask., in Regina, Sask., July 5, 1934.

BIRTHS

To DR. and MRS. ERNEST F. WALLER, University Farm, Saint Paul, Minn., a daughter, Leora Ann, June 19, 1934.

To DR. and MRS. C. H. PALS, of Chicago, Ill., a son, Calvan Herman, June 5, 1934.

To DR. and MRS. R. J. FLYNN, of Chelsea, Iowa, a daughter, Veronica Reed, June 19, 1934.

PERSONALS

DR. EDWARD E. THOMPSON (Iowa '34), has located at Woodstock, Va.

DR. ROGER M. COCKING (Iowa '34), may now be reached at Monona, Iowa.

DR. C. B. KRONE (K. S. C. '26) has removed from Clyde, Kan., to Concordia, same state.

DR. J. R. PORTEUS (N. Y. U. '18), formerly of Smyrna, Del., has removed to Trenton, N. J.

DR. W. C. KILPATRICK (Wash. '29), formerly of Yakima, Wash., has located at Colville, same state.

DR. CLARK A. WEAVER (O. S. U. '34) may now be addressed at 412 14th Street, Toledo, Ohio.

DR. A. WHITLOW (McK. '19), formerly of Swea City, Iowa, is now located at Armstrong, same state.

DR. H. H. SHEELER (Wash. '21), of Huntington Park, Calif., has purchased a home at 2427 Hope Street.

DR. WM. R. KERMEN (Wash. '34), of Cle Elum, Wash., has located at 932 Venice Blvd., Los Angeles, Calif.

DR. W. E. RASMUSSEN (Colo. '28), formerly of Ogden, Utah, is now located at Kaysville, same state.

DR. HARRY K. COPITHORN (U. P. '03) has moved from 58 Parkway, Chelsea, Mass., to Chatham, same state.

DR. E. A. CHAPMAN (Iowa '34) has selected Dodgeville, Wis., as a promising location for general practice.

DR. WALTER C. SNYDER (Corn. '18) has changed locations, from Watertown, N. Y., to Kenmore, same state.

DR. C. E. PETTEYS (McK. '17) may now be addressed at Grand Rapids, Ohio, instead of Weston, Ohio, as formerly.

DR. ARTHUR G. HALL (Corn. '08) has been transferred from Earlville, N. Y., to 110 Hudson Street, New York City.

DR. SAMUEL E. YOUNG (O. S. U. '17), of Pittsburgh, Pa., has retired from practice, according to a recent announcement.

DR. A. E. HANCOCK (O. S. U. '31), formerly of Williamsport, Ohio, is now located at 676 East Main Street, Lancaster, Ohio.

DR. BRUCE KESTER (O. S. U. '25) announces a change of address from Dayton, Ohio, to 1307 Summit Street, Lakewood, Ohio.

DR. E. W. WEBER (Wash. '20), of Alameda, Calif., suffered a paralytic stroke recently that has affected the use of his right side.

DR. JAMES R. BARRY (Iowa '29) has changed his address from Wonewoc, Wis., to 201 4th Avenue, N., South Saint Paul, Minn.

DR. HOWARD C. GALE (K. C. V. C. '10), of Concordia, Kan., has removed to Dallas, Tex. His new address is 2221 Cedar Springs.

DR. R. PAYNE MCCOMB (San Fran. '16), formerly of San Gabriel, Calif., is located at 532 East Haley Street, Santa Barbara, same state.

DR. PERCY M. ALDRICH (Wash. '32), who formerly received his mail at Dixie, Wash., may be reached at East 20 Sixth Avenue, Spokane, Wash.

DR. CHARLES GRUBER (Ind. '12), of Fort Wayne, Ind., last month rounded out 22 years as a railroad veterinarian and is still going strong.

DR. E. J. BATES (Mich. '25), formerly with the Michigan State Department of Agriculture, has located at Chesaning, Mich., for general practice.

DR. F. G. CASLICK (Corn. '30), of Morristown, N. J., recently moved his office from 4 Whippany Road to his residence at 159 Western Avenue.

DR. E. M. AUSTIN (Iowa '18) reports change of address from 203 Administration Bldg., Los Angeles Union Stock Yards, Los Angeles, Calif., to 314 Ojai Road, Santa Paula, same state.

DR. O. J. MAYFIELD (Iowa '28), who has been connected with the Food and Drug Administration, U. S. Department of Agriculture, at Beltsville, Md., has entered commercial work at Charles City, Iowa.

DR. J. I. NEAL (K. C. V. C. '15), of Sanford, N. C., is confined to the Pine Crest Manor, Southern Pines, N. C., with a pulmonary affection. He is reported to be making satisfactory progress toward recovery.

DR. ORVAL L. GATES (Colo. '25), formerly on the hospital staff of the American Society for the Prevention of Cruelty to Animals, New York City, has opened a hospital at 268 Broadway, East Paterson, N. J.

DR. W. T. OGLESBY (Iowa '31) has resigned his position in the Department of Physiology and Pharmacology at Michigan State College, to accept one in the Department of Veterinary Science at Louisiana State University.

DR. F. C. MECKSTROTH (Chi. '08), formerly of Saint Mary's, Ohio, has moved his family to Coldwater, Ohio, where he has taken over the practice of the late Dr. A. H. Dabbelt, whose death was reported in the April issue of the JOURNAL.

DR. W. AUSTIN EWALT (Ont. '07), of Mount Clemens, Mich., has opened a home for friendless animals on a 50-acre plot leased for the purpose near Mount Clemens. He will coöperate with welfare departments and humane societies in finding homes for dogs and cats.

DR. I. A. MERCHANT (Colo. '24), who has been on leave of absence from Iowa State College while taking work in public health at the Yale School of Medicine, New Haven, Conn., has returned to Ames to resume his duties in the Department of Veterinary Hygiene.

DR. WILLIAM J. PISTOR (Wash. '26) who has been acting professor of veterinary medicine at the State College of Washington for the past year, has resigned to accept a position at the University of Arizona, where he will head the Department of Animal Disease Research.